PB84-128032

# Mini-Assessment: Total Exposure Assessment and Exposure-Dose Relationships

ICF, Inc., Washington, DC

Prepared for

Environmental Protection Agency, Washington, DC

Dec 83

U.S. Department of Commerce
National Technical Information Service
NTIS

PB84-128032

EPA-600/6-84-002
December 1983

# MINI-ASSESSMENT: TOTAL EXPOSURE ASSESSMENT AND EXPOSURE-DOSE RELATIONSHIPS

by

William M. Mendez, Jr.
Kenneth B. Kolsky
Michael J. Gibbs

ICF Incorporated
1850 K Street, N.W.
Washington, D.C. 20006

Contract No. 68-02-3720

Project Officers

Marvin Rogul
Basil Manns

Office of Strategic Assessment and Special Studies
Office of Exploratory Research
Washington, DC 20460

OFFICE OF EXPLORATORY RESEARCH
OFFICE OF RESEARCH AND DEVELOPMENT
U.S. ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, DC 20460

REPRODUCED BY
NATIONAL TECHNICAL
INFORMATION SERVICE
U.S. DEPARTMENT OF COMMERCE
SPRINGFIELD, VA. 22161

## TECHNICAL REPORT DATA
*(Please read Instructions on the reverse before completing)*

| | | |
|---|---|---|
| 1. REPORT NO.<br>EPA-600/6-84-002 | 2. | 3. RECIPIENT'S ACCESSION NO.<br>PB8 4 128032 |
| 4. TITLE AND SUBTITLE<br>Mini-Assessment: Total Exposure Assessment and Exposure-Dose Relationships | | 5. REPORT DATE<br>December 1983 |
| | | 6. PERFORMING ORGANIZATION CODE |
| 7. AUTHOR(S)<br>William M. Mendez, Kenneth B. Kolsky, Michael C. Gibbs | | 8. PERFORMING ORGANIZATION REPORT NO. |
| 9. PERFORMING ORGANIZATION NAME AND ADDRESS<br>ICF Incorporated<br>1850 K Street, N.W.<br>Suite 950<br>Washington, D.C. 20006 | | 10. PROGRAM ELEMENT NO. |
| | | 11. CONTRACT/GRANT NO.<br>68-02-3720 |
| 12. SPONSORING AGENCY NAME AND ADDRESS<br>Office of Exploratory Research<br>Office of Research and Development<br>U.S. Environmental Protection Agency<br>Washington, D.C. 20460 | | 13. TYPE OF REPORT AND PERIOD COVERED<br>Final Report |
| | | 14. SPONSORING AGENCY CODE<br>EPA/600/20 |
| 15. SUPPLEMENTARY NOTES | | |

16. ABSTRACT

This report provides a brief examination of recent (1) research in the areas of total exposure assessment and (2) research concerning the relationship between patterns of exposure to environmental pollutants and dose levels received by exposed individuals and populations. This assessment is intended to provide policy makers with a brief, concise discussion of important trends in recent research; it is not meant to be a comprehensive literature review.

17. KEY WORDS AND DOCUMENT ANALYSIS

| a. DESCRIPTORS | b. IDENTIFIERS/OPEN ENDED TERMS | c. COSATI Field/Group |
|---|---|---|
| Exposure assessment, total exposure, risk assessment, monitoring, dose. | | |

| | | |
|---|---|---|
| 18. DISTRIBUTION STATEMENT<br>Unlimited | 19. SECURITY CLASS *(This Report)*<br>Unclassified | 21. NO. OF PAGES<br>158 |
| | 20. SECURITY CLASS *(This page)*<br>Unclassified | 22. PRICE |

EPA Form 2220-1 (9-73)

# NOTICE

This document has been reviewed in accordance with U.S. Environmental Protection Agency policy and approved for publication. Mention of trade names or commercial products does not constitute endorsement or recommendation for use.

# TABLE OF CONTENTS

PAGE

EXECUTIVE SUMMARY ES-1

1. INTRODUCTION 1-1

   1.1 The Assessment of Exposure and Dose 1-2

   1.2 Structure of the Report 1-5

2. MEASUREMENT OF INDIVIDUAL POLLUTANT EXPOSURES 2-1

   2.1 Introduction 2-1

   2.2 Framework for Measuring Individual Exposures 2-3

   2.3 Exposure Measurement Methods 2-7
      2.3.1 Introduction 2-7
      2.3.2 Personal Monitors 2-7
      2.3.3 Microenvironments 2-10

   2.4 Research Needs 2-16

3. ESTIMATION AND MEASUREMENT OF POPULATION EXPOSURES 3-1

   3.1 Introduction 3-1

   3.2 Methods for Estimating and Measuring Population Exposure 3-4
      3.2.1 Exposure Levels Based on Ambient Levels 3-4
      3.2.2 Use of Dispersion Models in Estimating Exposure Levels 3-7
      3.2.3 Population-at-Risk Estimates 3-10
      3.2.4 Recently Developed Exposure Assessment Methods 3-13

   3.3 Assessment of Total Population Exposures 3-17
      3.3.1 Introduction 3-17
      3.3.2 Total Exposure Models 3-19
      3.3.3 Total Exposure Applications 3-21

   3.4 Research Needs 3-26

## TABLE OF CONTENTS (cont'd)

PAGE

4. RELATIONSHIP BETWEEN PATTERNS OF EXPOSURE AND DOSE OF POLLUTANTS 4-1

  4.1 Introduction 4-1

  4.2 Relationships Between Exposure and Dose 4-2
    4.2.1 Properties of Pollutants Affecting Exposure-Dose Relationships 4-2
    4.2.2 Properties of Exposed Individuals and Populations Affecting Exposure-Dose Relationships 4-11
      -- Primary Demography Variables 4-12
      -- Socioeconomic Characteristics 4-15
      -- Activity Patterns and Physiological Variables 4-16

  4.3 Physiological Indicators of Dose 4-18

  4.4 Models for the Evaluation of Exposure-Dose Relationships and the Integration of Multisource Exposures 4-23

  4.5 Research Needs 4-29

List of References R-1

Total Exposure Annotated Bibliography B-1

# EXECUTIVE SUMMARY

This mini-assessment was commissioned by the Office of Strategic Assessment and Special Studies of the Office of Research and Development of the U.S. Environmental Protection Agency. It is intended to provide a brief examination of recent (1) research in the areas of total exposure assessment and (2) research concerning the relationship between patterns of exposure to environmental pollutants and dose levels received by exposed individuals and populations. This assessment is intended to provide policy makers with a brief, concise discussion of important trends in recent research; it is not meant to be a comprehensive literature review. The major focus is on the use of exposure assessment as a tool in the analysis of environmental policies, so emphasis will be placed on studies directly relevant to the assessment of such policies, rather than on more basic research. The annotated bibliography we have developed contains a number of references, however, which should enable the reader to obtain more in-depth information, if desired. Much of the research on which this mini-assessment is based was completed in August, 1982.

We also discuss possible areas where further research on total exposure assessment and exposure-dose relationships would be desirable. We developed this compilation of research priorities and areas where information is lacking from several sources: (1) the scientific literature and (2) personnel directly involved in research in exposure and exposure-dose assessment at EPA and other agencies of the federal government and in the academic and professional communities. In discussing possible research priorities we have emphasized, within certain limits, research needs most directly relevant to environmental policy assessments.

Chapter 1 of this report lays out a framework for evaluating research in exposure and dose assessment (Exhibit ES-1) which explains how the assessment of exposure and exposure-dose relationships influence the quality of environment risk assessment and where this research fits into the assessment of environmental policy. We point out how the study of the relationship between exposure and dose has often been neglected in the past and the nature of the assumptions that have had to be made in the absence of such information. The relatively recent appreciation of the importance of studies of total exposure (e.g., the identification of the important sources of pollutant exposure and the assessment of the relative importance of different routes of exposure for individuals and populations) is also discussed.

We also briefly review the important distinctions between population and individual exposure and demonstrate the various techniques for assessing or estimating each based on measurements or estimates of the other (see Exhibit ES-2). This distinction, along with the previously described framework for the use of exposure and dose assessment in environmental policy analysis provides the structure for the remainder of the report.

Chapter 2 is a discussion of techniques for the measurement and assessment of individual exposure to pollutants. Two different but complementary approaches to measuring total exposure to air pollutants, personal exposure monitoring and microenvironment characteristics, are discussed, and recent advances in personal monitoring devices are described. The relative strengths and limitations of sampling and monitoring devices for measuring exposures to specific pollutants are discussed and the current state of knowledge about microenvironmental characterization is reviewed. The bulk of this discussion deals with the characterization of indoor air quality.

## EXHIBIT ES-1
## FRAMEWORK FOR THE ANALYSIS OF POLLUTANT EXPOSURE

Activity (producing discharge of pollutant), rate of discharge for activity, activity levels for specific sources

↓

Discharge (release of pollutant from commercial control), models for environmental transport, transformation, distribution

↓

Presence (of pollutant in ambient environment: air, food water, etc., over time and in space), behavior of receptor, other modifying factors

↓

Exposure (interaction of pollutant and receptor), properties of pollutant, physiological state, demographic characteristics of exposed populations

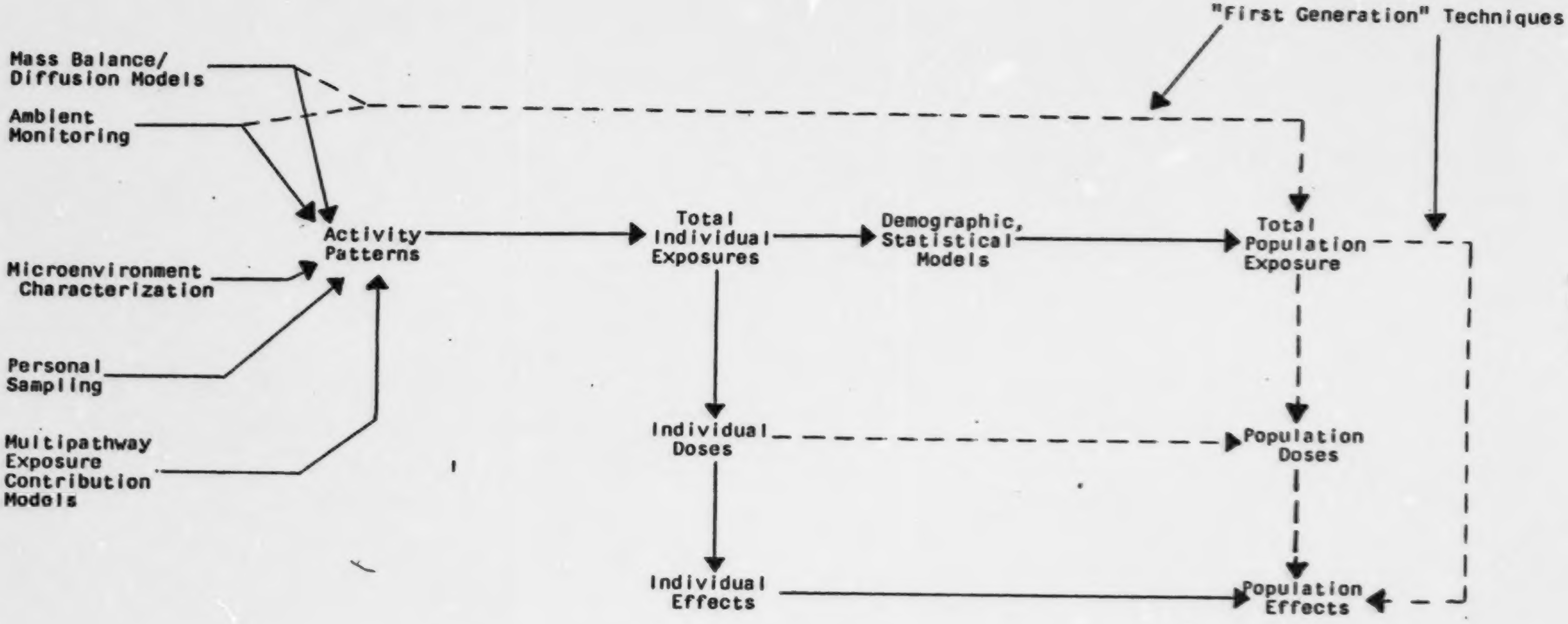
↓

Dose (amount of pollutant crossing boundary into receptor)

↓

Effects (on health or the environment)

### EXHIBIT ES-2
### ALTERNATIVE METHODS FOR ANALYZING POLLUTANT EXPOSURE AND DOSE LEVELS FOR INDIVIDUALS AND POPULATIONS


"First Generation" Techniques
Mass Balance/ Diffusion Models
Ambient Monitoring
Microenvironment Characterization
Personal Sampling
Multipathway Exposure Contribution Models
Activity Patterns
Total Individual Exposures
Demographic, Statistical Models
Total Population Exposure
Individual Doses
Population Doses
Individual Effects
Population Effects

Chapter 3 addresses methods for assessing and measuring population exposures to environmental pollutants both directly and by generalizing results from individual monitoring or through microenvironmental characterization. Exposure estimation techniques based on the use of ambient monitoring results are surveyed and the need for better models of human behavior is noted. Recent efforts to apply studies of population behavior (commuting or time use patterns, etc.) to the assessment of population exposures are discussed.

Chapter 4 deals with the determinants of relationships between exposure patterns and dose levels in pollutant-exposed individuals and populations. The chapter begins with a short review of recent studies of the properties of individual pollutants (solubility and particle size distribution, for example) which affect the dose absorbed by exposed individuals. This is followed by a discussion of how primary demographic variables (age, sex, race), socioeconomic factors (education, occupation, familial factors) and diseases or other abnormal physiological states can affect exposure-dose relationships for environmental pollutants. The chapter concludes with a discussion of mathematical models describing absorption and transport of pollutants in the body, models for integrating and comparing exposures by different routes and recent studies of physiologic indicators of pollutant exposure and dose and their potential applications in characterizing total exposures.

At the end of each chapter is a brief discussion of what we believe to be important information gaps and research needs. These findings can be summarized as follows:

## Chapter 2. Individual Exposure

- Improved personal monitoring devices for a number of pollutants (NOx, particulates) are required. Such devices need to be more sensitive, lighter, and capable of more or less continuous recording of pollutant levels if personnel exposures are to be characterized adequately.

- Improved sampling and analytical methods are needed for:

  - -- the characterization of trace organic contaminants in food; and metals and pesticides in individually prepared foods
  - -- better characterization of aerosols such as asbestos and particle-bound polycyclic hydrocarbons.

- continued studies to further characterize important pollutant microenvironments, when possible validated by personal sampling procedures, are needed.

## Chapter 3. Population Exposure

- EPA should develop a consensus on its exposure assessment needs and develop procedures to allow the most efficient use of resources devoted to exposure assessment.

- Existing data sources on environmental exposure (emission inventories, monitoring network results) need to be improved and updated to facilitate better characterization of population exposures.

- Specific improvements in study design are required including:

  - -- refinement of approaches to selection of population samples,

  - -- development of theoretical criteria for selection of microenvironments,

  - -- analysis of intercity and geographic variations in activity patterns and definition of microenvironments,

  - -- characterization of pollutant concentrations in microenvironments and variability over time,

  - development and validation of predictive exposure assessment models, and
  - analysis of alternatives for dealing with subjects regarding recording exposure dates, food intake, and activities during the day.

## Chapter 4. Exposure-Dose Relationships

- Attempts are needed to refine our understanding of the physiological variables affecting individual exposure-dose relationships in order to allow more accurate identification of high risk groups.
- Pharmacokinetic models of pollutant uptake and metabolism can be applied to the clarification of exposure-dose and dose-response relationships. More emphasis needs to be placed on assumptions regarding pollutant uptake as inputs to these models.,
- Continued efforts must be made to develop models which allow the assessment of contribution to total exposure from multiple sources and by multiple routes of exposure.

# 1. INTRODUCTION

Many human activities result in the release of potentially harmful substances into the environment. When these substances are found to be harmful to human health, to the environment or to result in aesthetically unacceptable conditions, they are classified as pollutants. Pollutants are generated both during production (e.g. air pollution from lead smelters) and consumption (e.g. particulates from automobile exhausts) activities.

Any societal action to control pollution (environmental regulation being a commonly used approach, but not necessarily the only approach) will involve tradeoffs between the positive effects of controlling pollution and the social costs associated with these control efforts. Clearly, careful and informed balancing of these factors is a necessary ingredient of any effective pollution control strategy, whether it be at the local, regional or national level.

The balancing of the costs and benefits of pollution control strategies requires an assessment of the effects of pollution on human health and the environment, both retrospectively, to obtain some idea of the extent of damage that has occurred, and prospectively, to predict the effectiveness of alternative strategies at controlling further harm. An integral part of the assessment of damage produced by environmental pollution (especially where chronic or subtle effects are involved or where direct epidemiological evidence is not available) is the measurement or estimation of the exposure to pollutants received by people and the doses which result from the exposures. Such data are required, in conjunction with the dose-response relationships derived from epidemiology, clinical studies, or animal studies, to assess the magnitude of damage to human health produced by pollution. The purpose of this mini-assessment is to provide an overview of the state-of-the-art in the assessment of human exposure to pollution, to review current research efforts in the area, and to identify areas where important gaps in information and theoretical knowledge have been found to exist.

## 1.1 THE ASSESSMENT OF EXPOSURE AND DOSE

Exhibit 1-1 summarizes a useful framework for the analysis of risks to human health caused by the release of pollutants into the environment.[1] It describes how substances are released into the environment, how they travel through the environment to the vicinity of human receptors, and how they interact with the exposed population to cause health effects. It paraphrases the steps that must be undertaken, at least implicitly, in any analysis of the effects of human exposure to environmental pollution. In each step of the analysis, information can be supplied either directly by observation or indirectly by the use of models and/or statistical inference. For example, exposure levels can either be measured directly for the population-at-risk using appropriate instruments and study designs, or they can be inferred based on measured levels of pollutants in the ambient environment, by taking into account the activity patterns, demographic characteristics and physiological states of the exposed populations as well as other factors affecting the levels of exposure received.

---

[1] Similar analytic structures have been developed by a number of authors. This particular framework is based on one developed by Ashford, et al., (1980).

## EXHIBIT 1-1
## FRAMEWORK FOR THE ANALYSIS OF POLLUTANT EXPOSURE

Activity (producing discharge of pollutant), rate of discharge for activity, activity levels for specific sources

↓

Discharge (release of pollutant from commercial control), models for environmental transport, transformation, distribution

↓

Presence (of pollutant in ambient environment: air, food water, etc., over time and in space), behavior of receptor, other modifying factors

↓

Exposure (interaction of pollutant and receptor), properties of pollutant, physiological state, demographic characteristics of exposed populations

↓

Dose (amount of pollutant crossing boundary into receptor)

↓

Effects (on health or the environment)

This report deals primarily with recent developments and current research efforts aimed at the direct measurement or estimation of either individual or population exposures to environmental pollutants. The focus of our discussion is on the development of exposure estimates as a tool for environmental policy analysis, rather than on exposure estimates as an end in themselves. Our discussion of research priorities and information gaps, while drawing heavily on research agendas developed by those working in the field of exposure assessment, also places such priorities within a broad, policy-oriented framework. For example, the performance of detailed market-basket surveys may be an important priority if one seeks precision in characterizing total exposure to food-borne pollutants for individuals. This may be a lower priority, however, if one is concerned with such assessments for large groups of people. This is because determining the average intake of pollutants for a population or population segment may be adequate, given other uncertainties in the analysis. Care will be taken to specify where such situations exist.

Placing a focus on exposure assessment as a tool for environmental policy analysis requires us to discuss recent research dealing with the relationship between measured and estimated exposures to pollutants and the dose of pollutants absorbed by exposed individuals and regulations. Throughout our discussion, we adopt the conventions suggested by Ott (1980), defining exposure as the amount of pollutant impinging upon the surface (skin or mucous membranes) of exposed individuals over time, and dose as the amount of pollutant actually absorbed into the body of those individuals. We define "total exposure" to mean the characterization of all of the sources of exposure to pollutants experienced by a person or a population. Thus, a major focus of this paper is on multi-source and multi-pollutant exposures.

The relationship between exposure and absorbed dose is important in assessing the significance for human health of exposure to toxic pollutants. The complex relationship between exposure and dose and its significance for analyzing the health effects of pollutant exposure have become widely recognized. This relationship is especially important in the area of toxic metals pollution and for other chronic and cumulative toxins where differences in route of exposure, chemical and physical form of the substance in question, and physiological state of the exposed population can all have an important bearing upon the dose resulting from a given exposure.

## 1.2 STRUCTURE OF THE REPORT

The remainder of this report is divided into three chapters, corresponding to important areas of research in total exposure and dose assessment. Chapter 2 addresses techniques for measuring and estimating the exposure of individuals to pollutants. Included in this chapter are discussions of the relationship between ambient pollutant levels and individual exposures and techniques for directly measuring individual exposure to pollutants. In addition we will discuss recent study results where individual exposures to pollutants have been investigated and attempt to identify research needs and information gaps in the measurement and estimation of total individual exposures.

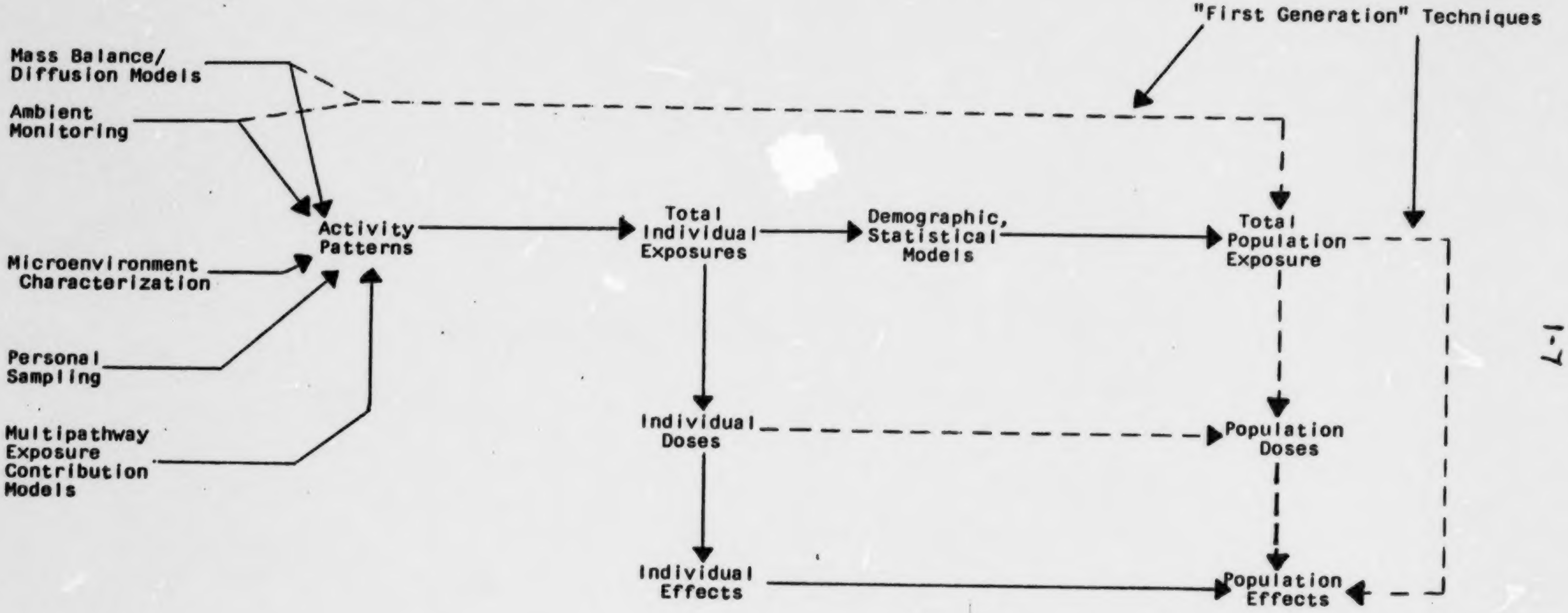
The relationship between ambient pollutant levels in the environment and individual exposure is important. Often much of the information available for estimating individual exposure comes from monitoring systems which measure ambient concentrations of pollutant in one or another medium or from mass-balance and diffusion modeling of pollutant releases. Exhibit 1-2 illustrates the alternative methods for analyzing pollutant exposure and dose for individuals and populations.

To develop a more accurate picture of the exposure of any individual to a pollutant, it is necessary to take into account the activity patterns of that individual. This allows determination of how the concentration of pollutants in the ambient environment (air, food, water, etc.) contributes to the total exposure received. We therefore discuss the current practices in relating ambient pollutant levels to individual exposure including simple models for taking activity patterns (eating, smoking, mobility, etc.) into account. We also discuss recent advances in techniques by which individual exposures to pollutants can be assessed directly.

In Chapter 2 we review the more comprehensive models that have recently been developed for incorporating individual sampling data and ambient pollutant monitoring results to produce a more complete picture of individual exposures to environmental pollutants. The major efforts in this direction have included attempts to characterize pollutant exposures associated with "microenvironments", with much effort in this area directed toward previously unstudied indoor pollutant exposures. Results of recent research are briefly reviewed, with emphasis on studies which have attempted to estimate the relative contributions of multiple sources of a single pollutant to individual exposures and to quantify total individual exposure to a number of different pollutants. Chapter 2 ends with a brief discussion of research needs and information gaps from the point of view of risk analysis.

Chapter 3 discusses the estimation and measurement of population exposures. Estimating population exposure is relatively simple in a situation where everyone exposed to a pollutant encounters the same levels of pollutant

## EXHIBIT 1-2
## ALTERNATIVE METHODS FOR ANALYZING POLLUTANT EXPOSURE AND DOSE LEVELS FOR INDIVIDUALS AND POPULATIONS


"First Generation" Techniques
Mass Balance/ Diffusion Models
Ambient Monitoring
Microenvironment Characterization
Personal Sampling
Multipathway Exposure Contribution Models
Activity Patterns
Total Individual Exposures
Demographic, Statistical Models
Total Population Exposure
Individual Doses
Population Doses
Individual Effects
Population Effects

in the ambient environment and undertakes the same kinds of activities which lead to exposures. In this situation everyone will receive roughly the same exposure. More frequently, however, available data concerning individual exposure or ambient pollutant levels are relevant only to one small subset of the exposed population or to only a particular geographic location. A typical example might be an attempt to characterize population exposure to volatile organic chemicals (VOC) in a metropolitan area based on monitoring data from an outdoor air sampling network in the area, levels of VOC's in food and water, and personal air and body burden sampling data from one particular group of people living in the area. In this case, methods would need to be found to incorporate all of the data about the distribution of pollution and the exposed population and for analyzing differences in activity patterns of various groups of individuals that modify exposures relative to measured values.

Chapter 3 begins with a brief review of "first generation" methods for estimating and measuring population exposure. These studies are of interest because they represent the scientific bases for many regulations controlling environmental pollution, and refinements in these studies point to corresponding refinements in programs to control pollution.

Chapter 3 also explores methods which have been developed more recently to characterize population exposure to pollutants. Many of these methodologies are being developed in parallel with the methodologies previously described for estimating individual exposure. Important areas of discussion include the application of microenvironment characterization studies for the estimation of population exposure and assessment of population total exposures to multimedia pollutants. The chapter concludes with a review of research needs.

Chapter 4 discusses the relationship between exposure and the dose of pollutant received by exposed individuals and populations. This area has only recently received research attention. The dose of pollutant absorbed is an important determinant of the magnitude of health effect associated with a particular exposure and there are many factors--such as the physical form of the pollutant and the physiological state, age and activity patterns of the exposed population--which can alter the actual dose received by individuals.

Chapter 4 also reviews the properties of pollutants, such as the size distribution of particulates which are known to influence exposure-dose relationships. Socioeconomic and demographic variables which affect these relationships are also dealt with. We then briefly review the current research on the use of physiological indicators of exposure and dose and review recently developed models for relating exposure, dose and effect of pollutants from single and multiple sources.

# 2. MEASUREMENT OF INDIVIDUAL POLLUTANT EXPOSURES

## 2.1 INTRODUCTION

This chapter discusses the methods available for measuring the exposure of individuals to pollutants in air, water, and food. Such exposure measurements are fundamental pieces of information required to characterize total exposures and undertake societal actions to control pollution in an efficient manner. Without measurements or estimates of individual pollutant exposures, the balancing of costs and benefits of various control options (one of which may be regulation) cannot be made. At a minimum, measurements of individual pollutant exposures can be used to calibrate and/or validate population exposure estimates derived by various methods.[1] Additionally, individual exposure measurements can be useful for identifying the relative importance of various sources of a given pollutant, and for enhancing the power of epidemiologic studies to identify the consequences of exposure (Yanagisawa and Nishimura, 1982).

The characterization of individual exposures to environmental pollutants is a fairly recent undertaking. Prior to 1970, personal exposure monitors for air pollutants (monitoring devices that could be carried by individuals as they went about their daily routines) were not available (Ott and Flachsbart, 1982). Air pollution exposures could only be measured through the use of either portable equipment for filling sample bags, and then transporting the samples to a laboratory for analysis, or with larger stationary sampling stations. Similarly, water quality has been measured primarily at fixed monitoring stations (EPA, 1981f), and methods for analyzing many chemicals in foods were (and are) not available (EPA, 1981b). Several studies have demonstrated that existing ambient monitoring does not reflect actual exposures to various air pollutants such as CO (Ott, 1971; Cortese, 1976; Wallace, 1979), $NO_2$ (Nitta and Maeda, 1982), and VOC (Zweidinger, et al., 1981). Therefore, ambient monitoring data may be viewed as at least somewhat suspect for purposes of individual exposure evaluation, and should be used with caution. In some cases, the characterization of the populations-at-risk (discussed in Chapter 3) could be improved with individual exposure data, and in almost all cases, the evaluation of individual risk requires individual exposure information.

---

[1] Measurement and estimation of population exposure is discuss in Chapter 3.

Significant progress has been made over the past decade, both conceptually and instrumentationally, for evaluating individual exposures. A comprehensive framework for analyzing individual exposure to air pollutants was put forward by Ott (1980) that incorporates the notions of concentration, duration, and variability. This framework for evaluating individual exposures is first discussed briefly in Section 2.2 in order to identify the data necessary to implement the framework and evaluate individual exposures. Next, in Section 2.3 the methods and instruments that have been developed over the past decade to collect the necessary data are reviewed, and results of recent studies of individual exposure are presented. Finally, the research needed to improve the characterization of individual exposures is identified in Section 2.4.

## 2.2 FRAMEWORK FOR MEASURING INDIVIDUAL EXPOSURES[2]

Individual exposure is defined here as the intersection of the location of a person with the location of a given concentration of pollutant, at a given time. Exposure, therefore, has both spacial and temporal significance. The units of exposure may vary, depending on the method and purpose of the exposure calculation. The instantaneous exposure of an individual would be measured as a concentration (e.g., ppm). For example, the concentration of CO in the air at a particular location at a given time may be 2 ppm. To describe the exposure of an individual over a period of time (e.g., 24 hours), the integrated exposure is required. The integrated exposure is calculated by multiplying the instantaneous exposure by its duration. For example, in a 24-hour period an individual may be exposed to nine hours of CO at 2 ppm while at work, two hours of CO at 15 ppm while in transit, and 13 hours of CO at 1 ppm while at home. The individual's integrated exposure over the 24-hour period would be (9x2) + (2x15) + (13x1)= 61 ppm-hrs. of integrated exposure. The person's average exposure would be 61 ppm-hrs. ÷ 24 hrs. = 2.54 ppm. A more complete picture of the individual's exposure could be represented by a graph which shows his exposure concentration throughout the entire 24-hour period (see Exhibit 2-1). Additionally, running averages could be calculated for given time intervals (e.g., 8 hours) which may have relevance for health risk assessment and regulatory design.

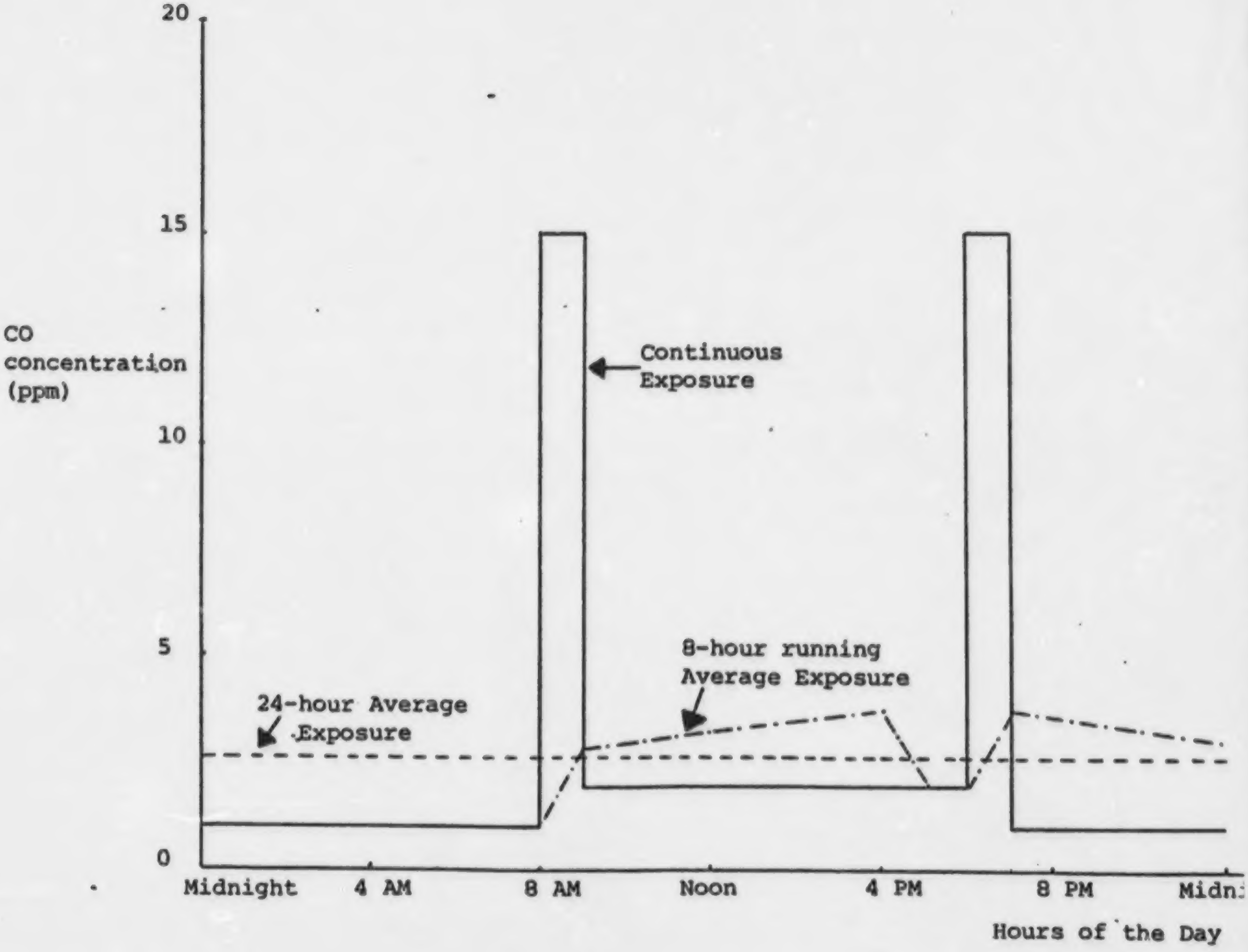
The most appropriate representation of an individual's exposure depends primarily on the anticipated use of the measurements. To determine an individual's peak exposure, the entire graph of instantaneous exposure shown

---

[2] This section draws heavily on Ott, (1980).

# EXHIBIT 2-1

## HYPOTHETICAL GRAPH SHOWING THE VARIABILITY OF AN INDIVIDUAL'S EXPOSURE TO CO DURING A 24-HOUR PERIOD



in Exhibit 2-1 would be required. The 24-hour average exposure for the individual could be represented much more simply as a single number. The fundamental question for research planning in the area of individual exposure measurement is to what extent the entire time profile of exposure shown in Exhibit 2-1 is required, and under what circumstances is the 24-hour (or some other time) average adequate? The answer to this question has important implications for the instrumentation and methods required to supply the exposure information.

The entire time profile of an individual's exposure is useful for several closely related purposes. The time profile aids in the identification of sources of pollution. High pollution exposure concentrations during certain times of the day can be associated with certain activities. For example, exposure to CO during transit accounts for nearly 50 percent of the integrated CO exposure shown in the hypothetical graph in Exhibit 2-1. The 24-hour average exposure of 2.54 ppm does not convey this fact. Therefore, the full exposure pattern is useful for identifying those control options which would be most effective in reducing the exposure of the individual.[3]

A second use for the entire time profile of exposure is the identification of relatively homogeneous microenvironments. A microenvironment is defined as a location or class of locations which can be treated as identical from the standpoint of pollutant exposure (EPA, 1981e). The three microenvironments in the above example are home, work, and transit. Each microenvironment has a unique concentration associated with it, and the exposure of any individual can be characterized by the amount of time he spends in each microenvironment. The advantage of identifying a limited number of mutually exclusive and exhaustive microenvironments is that the microenvironments can be monitored and characterized using portable monitoring equipment which need not be as intrusive as personal monitors which people carry with them during their daily routine. Moreover, the identification and characterization of microenvironments can enhance the ability to estimate individual total exposures without taking a personal sample for each individual. Only activity patterns among microenvironments need to be obtained.

---

[3] The shapes of the exposure-dose and dose-response relationships for the pollutant are also of great importance in determining the effectiveness of various control options in reducing the effects of exposures. The importance of time profile exposure estimates in the analysis of these relationships is discussed further in Chapters 3 and 4.

Finally, the time profile of individual exposures is useful for putting exposure estimates into units comparable to those used for describing risk. For example, the risks from exposure to CO may be expressed as a function of an 8-hour average exposure. The time profile of exposure allows one to calculate running 8-hour average exposures as shown in Exhibit 2-1.

It should be noted that although the above example relates to exposure to air pollution, this same framework can apply to exposure to contaminants in water and food. Exposures from water and food can have important time dependent elements. Water exposures can also be broken down into dermal and ingestion; and foods similarly can be divided along lines such as fresh produce, fish, processed foods, etc. Therefore, this framework can be generalized to include all sources of exposure so that the total exposure of an individual can be estimated.

Clearly, then, there are important uses for the detailed time profile of exposures. The next section discusses the methods and instruments available and required for obtaining the data needed to implement this exposure framework.

## 2.3 EXPOSURE MEASUREMENT METHODS

### 2.3.1 Introduction

There are two basic approaches for estimating individual exposures: (1) sampling an individual's exposure with a personal monitoring device; and (2) sampling microenvironments and then estimating the individual's exposure based on his activity pattern among the microenvironments.[4] As mentioned above, it is useful to coordinate the use of these two approaches. Personal monitoring devices can be used to identify potential microenvironments; which could be analyzed and subsequently used to estimate the exposures of an individual with a known activity pattern. Therefore, once microenvironments are defined and analyzed, an individual's exposure can be determined from his activity pattern, and a personal monitor is not required.

Personal monitors and microenvironments are discussed below. Special attention is paid to indoor air quality in the discussion of microenvironments because people spend the majority of their time indoors.

### 2.3.2 Personal Monitors

Personal monitors refer primarily to small devices worn or carried by an individual that can be used to measure the exposure of the individual to a given air pollutant. Because personal monitors are not applicable for measuring exposure to pollutants in water or food, the following discussion relates exclusively to air pollution. Techniques for sampling and analyzing water and food are discussed in the next section.

---

[4] Ambient monitoring is a generalized case of the microenvironment approach. However, the "microenvironment" as defined by ambient monitoring has been found to be too large and too nonhomogeneous to be of practical use in estimating individual exposures. Therefore, ambient monitoring is not considered here as a means of estimating individual exposures.

Over the past five years major advances have been made that for the first time make possible accurate and easy measurement of an individual's exposure to various air pollutants (Wallace and Ott, 1982). A variety of personal monitors were recently reviewed at the International Symposium on Indoor Air Pollution, Health and Energy Conservation (Harvard University, October 13-16, 1981) and by Wallace and Ott (1982), and much of this discussion is drawn from these sources.

Personal monitors are currently available or are reportedly in various stages of development for the following substances:

- Carbon Monoxide (CO);
- Nitrogen Dioxide ($NO_2$);
- Respirable suspended particulates (RSP) including nitrates, sulfates and metals;
- Some (nonpolar) volatile organics;
- Organochlorine pesticides and polychlorinated biphenyls (PCB); and
- Polynuclear aromatics.

The personal monitors operate in a variety of ways; the major difference across personal monitors being that some are analytical and others are essentially sampling devices.

Analytical personal monitors are able to measure the concentration of the pollutant at a given time. For example, two CO monitors (the ESI ECOlyzer

9000 and the GE CO "Detector") are capable of supplying "instantaneous" CO concentrations. Sampling or collection devices, on the other hand, capture the pollutant over a given sampling period (e.g., 8 hours), and the sample is subsequently analyzed in the laboratory. An example of a collection type personal monitor is the Palmes $NO_2$ diffusion tube (Wallace and Ott, 1982).

An analytical monitor provides a time profile of exposure which can be related to individual activities. This information can be used to identify sources of contamination and/or potential microenvironments. Only the CO personal monitors mentioned above are of this type.[5]

The collection monitors can only provide integrated exposure estimates over the entire sampling period. For example, the Palmes $NO_2$ diffusion tube requires rather long sampling periods (several days) for measurement of levels of $NO_2$ commonly occurring in ambient air (Yanagisawa and Nishimura, 1982). The resulting exposure estimates (in ppm-hrs or average ppm) do not provide as much information as analytical monitors would, and may not be as useful for identifying microenvironments and sources of pollution. However, even integrated exposure estimates have been found to be major improvements over ambient monitoring data for the purpose of estimating individual exposures. Furthermore, once a microenvironment has been identified, these devices are useful for evaluation purposes.

The shorter the sampling period required for collection-type personal monitors, the closer they come to providing time profile exposure estimates.

---

[5] A portable analytical monitor capable of estimating the mass of RSP (but not the chemical composition) has also been developed. This device is too large to be considered a personal monitor. See Wallace and Ott (1982).

A $NO_2$ sampler developed in Japan may allow measurements over periods as short as from four to eight hours. An individual could use a series of such sampling devices to sketch out a rough time profile of exposure.

No large scale use has been made of personal monitors to date, and only several smaller studies have been undertaken. Work has centered primarily on the development and testing of individual devices, and problems associated with the logistics of field studies involving a large number of people remain to be addressed. However, results of several pilot studies of uses of personal samplers are not only interesting in and of themselves, but they also indicate the large potential value of more widespread application of these devices. For example, in 1980, EPA conducted a small study of personal exposure to volatile organic compounds (VOC) at two locations---Lamar University, Beaumont, TX, and the University of North Carolina, Chapel Hill, N.C. (Zweidinger, et al., 1981). Research subjects carried personal monitors for 5 to 9 hour periods during their daily routine. The study not only indicated the feasibility of measuring individual exposures to VOC, but also demonstrated that there is a wide variability of exposures among a homogeneous group of subjects in a small area.

Personal monitors have been used somewhat more extensively in the identification and evaluation of microenvironments. This concept and the progress made to date in evaluating microenvironments is discussed next.

### 2.3.3 Microenvironments

A microenvironment is a location, or class of locations, which can be treated as identical from the standpoint of pollutant exposure (EPA, 1981e).

The identification and evaluation of microenvironments is valuable for estimating individual exposures to environmental contaminants. This section describes the progress that has been made in the characterization of microenvironments, and the next chapter discusses how microenvironment data can be used to estimate population exposures.

**Microenvironments for Indoor Air.** Numerous studies have been undertaken to characterize indoor areas as microenvironments. In nearly all cases, indoor air has been found to be markedly different from outdoor air (Moschandreas, 1978; Yocom, 1982). Furthermore, the examination of indoor air has led to the identification of significant indoor sources of pollution in residences and commercial spaces.

Yocum (1982) has recently reviewed most of the available assessments of indoor air and has drawn the following conclusions about individual indoor exposures relative to outdoor levels:

- Sulfur Dioxide: Outdoor concentrations may overpredict indoor exposure by a factor of about 2;
- Total Particulate Matter or Respirable Particulate Matter: Indoor exposures bear little relationship to outdoor concentration patterns. Additionally, the chemical and physical nature of indoor particulate matter is different from outdoors.
- Carbon Monoxide: Indoor sources of CO cause indoor exposures to be greater than outdoor exposures. However, in the absence of indoor sources (such as stoves or heaters) long run outdoor averages are reasonably good predictors of indoor exposure.
- Nitrogen Dioxide: Indoor sources of $NO_2$ (like those of CO) cause indoor exposures to be greater than outdoor.
- Nonmethane Hydrocarbons: There are numerous indoor sources of hydrocarbons and other organic gases and vapors such that outdoor concentrations under-predict indoor exposures by a factor of about 1.5.

- Lead: There are no significant indoor sources of airborne lead, so that outdoor concentrations over-predict indoor exposures.
- Sulfates: Outdoor concentrations tend to exceed indoor exposures by a factor of about 1.5.
- Radon and Formaldehyde: These are both of little importance in outdoor air, but have been found to have important indoor sources.

Although these conclusions presented by Yocum provide a reasonably good picture of the indoor concentration of several important pollutants relative to outdoor concentrations, additional data are required for some substances in order to distinguish adequately among different types of indoor microenvironments. For example, Ott and Flachsbart (1982) report using personal CO monitors to characterize the air quality in indoor commercial spaces. (Commercial settings were defined as locations in which the general public conducts routine business or to which it has access; e.g., stores, banks, offices, hotels, theaters, restaurants, etc.). Their results indicate that commercial spaces can be divided into two types: (1) those with indoor parking facilities attached; and (2) all others. Buildings with indoor parking facilities had markedly higher CO concentrations, in some cases on many floors in the building. Other commercial spaces (without parking facilities) consistently had lower CO concentrations (approximately 1 to 5 ppm) as a group. Even this characterization of two indoor commercial microenvironments for CO could (and possibly should) be further specified to include time as a factor (EPA, 1981e). Although Ott and Flachsbart found fairly constant concentrations at individual locations for periods as long as 111 minutes, it is likely that concentrations would vary significantly with time of day (e.g., evening rush hour as opposed to late evening).

Another example of the identification of indoor microenvironments is the evaluations of indoor residences undertaken by Moschandreas (1978). In that study, monitors for a variety of pollutants were placed in three locations in the home (kitchen, bedroom, living room) for fourteen days. Analysis of the measurements indicated that there were only very slight differences in the concentrations measured by the three monitors. Moschandreas concluded that a one-zone representation of indoor residences was adequate so that one indoor hourly average concentration per pollutant could be used to characterize the residential air quality for that hour. Therefore, it may be that many or most individual homes need not be subdivided into smaller microenvironments.[6]

Moschandreas did find, however, that residences should be divided into two broad classes:

- those with indoor pollution sources (such as gas appliances, smoking, cleaning activities, cooking activities); and
- those without indoor pollution sources.

Gas-fired stoves and furnaces were found to contribute significantly to concentrations of CO, NO, and $NO_2$. These results have been confirmed by Good, _et al_. (1982), Nitta and Maeda (1982), Colome, _et al_. (1981) and others. As a contrast, Moschandreas found no significant indoor sources of ozone, so that all residents could be grouped together into the same microenvironment for ozone.

---

[6] cf. Yocum (1982), p.509

Clearly, then, a variety of indoor microenvironments can be developed for each pollutant of interest. The following indoor microenvironments have been put forth for CO (EPA, 1981e):

- Parking garages;
- Buildings with attached parking garages;
- Indoor commercial settings (on congested streets);
- Residences with all electric appliances;
- Residences with gas appliances (stoves and heaters, vented and unvented); and
- Indoor locations with heavy smoking.

Comprehensive indoor microenvironments for other air pollutants have not been identified.

**Other Microenvironments**. Several other microenvironments have been examined and characterized to various degrees. The availability of easy to use monitors for CO has allowed numerous investigations of CO exposures in various locations such as cars, buses, city streets, etc. Colwill and Hickman (1980) reported CO levels between 12 and 60 ppm inside 11 new small cars driven around a 35 kilometer route in and around London. These levels were between 30 and 80 percent of the external concentrations found, and were noted to depend on the ventilation systems in the different cars.

In Boston, CO concentrations experienced by commuters during trips lasting 45 to 60 minutes averaged 11.9 ppm, compared to 6 ppm observed at six fixed air monitoring stations (EPA, 1981e). In Toronto, similar results were reported for pedestrians and street workers whose exposures were found to be between 10 and 50 ppm. In Washington, D.C., the average CO exposure for several car and bus passengers was found to be 11.7 ppm ± 4.9 ppm, and air-conditioned buses reportedly had levels 60 percent higher than non-air-conditioned buses (Wallace, 1979). Other studies have examined the exposure of bicyclists and traffic control personnel (Jabara, et al., 1980) as well as other individuals in suburban and urban areas.

Patterns of exposure to other air pollutants have been much less studied. The 1980 EPA study of VOC exposures at Lamar University (Beaumont, TX) and the University of North Carolina (Chapel Hill, N.C.) mentioned above found that exposures varied considerably across individuals within each location, but not across the two locations. This resulted despite the large concentration of petrochemical production near Beaumont, TX. The variances across individuals were not accounted for, and microenvironments were not defined.[7]

Several preliminary studies of exposure to respirable suspended particulates (RSP) have been made using a portable monitor capable of determining the total mass of particulates (Wallace and Ott, 1982). This device, called a piezobalance, has been used to examine the contribution of tobacco smoke to the concentration of RSP in various locations.

Even fewer studies have been performed to examine individual exposures to pollutants in water and food. Techniques are available for measuring many chemicals in water, however, there are no validated protocols for analyzing individual food samples for low levels of contaminants.

EPA's recent TEAM Study analyses demonstrate that drinking water can be a significant contributor of several organic compounds to an individual's total exposure; most notably chloroform and bromodichloromethane. Other organics were found in water, but at concentrations leading to daily intakes at least one order of magnitude below those found in air. Of course total dose (as opposed to total exposure) is the more important concern from a health effects standpoint. Because exposure-dose relationships will vary across contaminants and across media, the relative importance of air and water concentrations of these organic compounds cannot be determined from exposure estimates alone.

---

[7] It should be noted that the identification and characterization of microenvironments was not an objective of this EPA study. Nonetheless, the study results indicate that there may be important VOC microenvironments which account for the variances of exposures among the research subjects.

Recently, FDA has had some success with analyzing volatile organics in beverages, oils, and fish, but methods are lacking for numerous chemicals in different food groups (EPA, 1981f). EPA recently utilized these methods in the TEAM study (the tests were performed at an FDA laboratory). With the possible exception of trichloroethylene, food was found to be an insignificant source of exposure to volatile organics.

## 2.4 RESEARCH NEEDS

In order to obtain the necessary data to evaluate individual exposure within the framework outlined above, a variety of research is required. Several reports and papers on research needs in this general area have been prepared (EPA, 1981f; EPA, 1981b; EPA, 1981c; Wallace and Ott, 1982), and a variety of recommendations have been made regarding the needs for:

- improved personal and portable monitoring devices for air pollutants;
- better sampling procedures;
- analytical techniques capable of identifying very low concentrations of pollutants;
- protocols for analyzing individually prepared food samples; and
- the identification and characterization of microenvironments.

These different needs are closely related. For example, the development of advanced personal monitors for CO led to the collection of very valuable data regarding the variability of CO exposures across microenvironments. Considerable work remains to be done on developing a comprehensive list of microenvironments for CO, but it has been demonstrated that the microenvironment concept can be implemented to estimate individual exposures to CO in air. Without the advanced personal monitors for CO, much of this progress could not have been made as rapidly as it was.

Although this reasoning implies that advanced personal monitors similar to those currently available for CO are desirable for a large number of pollutants, it is not at all clear that such a goal is feasible or cost-effective. There are scores (if not hundreds) of compounds of sufficient potential health significance so that detailed exposure profiles may be desired for each. Given current and future resource availability, it is unrealistic to expect that such a massive undertaking could be feasible. Therefore, a comprehensive examination of priorities across both pollutants and media is required.

Several important criteria for setting research priorities in this area may include:

- the prevalence of the pollutant;
- the health significance of the pollutant;
- current understanding of the sources of the pollutant; and
- current understanding of the transport phenomena of the pollutant.

Those pollutants that are most ubiquitous, of greatest health significance, with least understood sources and transport phenomena would be given highest priority for the development of personal monitors. Not surprisingly, several criteria air pollutants (particulates, CO, $NO_2$) that are very ubiquitous and of health significance have also generated considerable personal monitor research activity. As these monitors become perfected, studies similar to those performed for CO should be undertaken.

Of greatest importance for most other substances in air, water, and food is the development of sampling and analytic techniques that will permit the detection of low concentrations over short sampling periods (e.g., eight or fewer hours). Methods are lacking for a variety of important substances including:

- non-volatile and volatile organics in food;
- aerosols, asbestos, and polyaromatic hydrocarbons in air; and
- analysis of particulate organics in air.

Techniques are apparently available for most important compounds found in water. However, much remains to be done regarding the actual exposure and dose levels of individuals to these compounds through ingestion or dermal contact.

# 3. ESTIMATION AND MEASUREMENT OF POPULATION EXPOSURES

## 3.1 INTRODUCTION

A critical factor in estimating the population risk of harm from environmental pollutants is the estimate of the population exposure to the pollutants (see Exhibit 1-1). Many authorities agree that the weakest link in developing estimates of population risk is the lack of knowledge on exposure (EPA, 1981f; EPA, 1981e). EPA does not possess a scientifically defensible, peer-reviewed methodology for projecting future exposures of the population, or a scientifically validated technique for estimating changes in exposures in response to different regulatory and emission control strategies.

Exposure assessment involves estimates of the size of the exposed population and the concentration levels and durations to which it is exposed. Exposure calculations should be made consistent with the requirements of the dose-response relationship that may later be used in a risk assessment. Examples of such requirements are annual average exposures, peak exposures, exposures that are greater than some threshold value, or the frequency and distribution of intermittent exposures. The dose-response relationship may require linear or non-linear and continuous or non-continuous functions of these exposure measures.

Reversible health effects may be associated with exposure below some "threshold" concentration level. Below this level, the body can repair damage rapidly enough to suppress symptoms and prevent permanent harm. Above this level, the human body becomes less capable of repairing damage at a satisfactory rate. Therefore, symptoms appear in more and more people in the exposed population. The dose-response function for pollutants of this type are characterized by a threshold concentration or integrated exposure below which no damage is experienced. Research efforts have found widely varying thresholds for different pollutants (Rydell, et al., 1968).

Some pollutants cause irreversible effects even at low concentrations. The total damage to the body may be related to the total pollutant dosage the body receives. Some bodily damage may be reversible at low levels of exposure but irreversible at higher levels. Risk assessments for carcinogens, for example, are generally based on linear or multi-hit models which do not include a threshold. Estimation of individual exposure at various levels of population exposure may not be necessary for purposes of these risk assessments because the total health risk would be proportional to the long-term exposure summed for all affected people over the period covered by the analysis.[1] Exposure of many people at low concentrations would produce the same number of cancers as would exposure of a few people to higher concentrations. The risk assessment could therefore be based on aggregate population exposure levels. (Measurement of individual exposures might still be necessary to validate estimates of population exposure.)

Risk assessment models also often assume continuous exposure to a pollutant. Some exposures however are intermittent and there is evidence that the damage function is modified for these exposure situations or when exposure to a pollutant occurs in combination with other pollutants (Rydell, et al., 1968). With intermittent exposures, the body may repair itself between exposures. Some "safe" exposure limits are based on specific patterns of exposure such as those patterns faced by workers during a regular 8-hour per day, 5-day work week. Adjustments to the "safe" limits must be made when workers work overtime or also face environmental exposures to the same pollutants (Hickey and Reist, 1979). In these cases, it is important that the pattern of exposure be studied and not simply the pollutant dose received.

---

[1] This holds not only for linear models, but for most multistage models at low concentrations where population risk is a linear function of dose and exposure.

It can be seen that in analyzing the population's exposure to a pollutant, one should have an idea of the form of the dose-response model to be used in the subsequent risk assessment. The lack of good exposure data however often leads to the adoption of non-threshold, linear models, and for pollutants where non-linearity in dose-response relationships is expected, data requirements are more extensive and aggregate exposure estimates will not suffice.

The remainder of this chapter is divided into three sections. Section 3.2 reviews the methods used to estimate population exposure to a given environmental pollutant. These methods have frequently been based on use of ambient levels of pollutants measured by fixed monitoring stations. Recent studies have pointed out the shortcomings of this approach and new methods are emerging. Section 3.3 reviews the issues involved and past efforts directed at measuring the population's total exposure to a pollutant or group of pollutants from multiple sources or media. The main routes for human exposure are air, water, and food. Concurrent measurement of exposure from these routes will contribute to a better characterization of how total exposures would vary under different exposure conditions and will help identify the best control approaches. While the need for a total exposure approach to analysis of health effects of environmental pollutants seems clear; organizational, resource, and technical problems have slowed the development of this approach. Chapter 3 concludes with a review in Section 3.4 of research needs regarding estimation and measurement of population exposure. Fulfillment of these needs should make an important contribution to improved population exposure estimates and therefore to more soundly based decisions regarding protection of human health from environmental pollutants.

## 3.2 METHODS FOR ESTIMATING AND MEASURING POPULATION EXPOSURE

Population exposure estimates require estimates of the levels of exposure and the numbers of people exposed to those levels. Many past exposure assessments for a particular pollutant have based exposure level estimates on measured ambient levels for that pollutant and numbers of people exposed estimates on available population density data. In section 3.2.1 we review the soundness of an approach involving estimating population exposure levels based on ambient monitoring data. In Section 3.2.2 we discuss another common approach to estimation of population exposure levels -- the use of dispersion modeling. Section 3.2.3 reviews the methods that have been used to estimate the population at risk for pollutants. Section 3.2.4 then discusses some of the more recent exposure assessment methods.

### 3.2.1 Exposure Levels Based on Ambient Levels

In the past decade, a number of studies have attempted to estimate exposures by using data from fixed air monitoring stations. The extent to which fixed stations give values that are representative of the exposures of the population of an area is not known accurately (Ott, 1981). However, data from fixed air monitoring stations often have been demonstrated to differ from the concentrations with which people actually come into contact. Therefore, these approaches give exposure estimates of unknown precision and questionable quality.

Recent studies indicate that outdoor fixed-station readings do not meaningfully reflect total human exposure to any of the following pollutant classes: respirable particulates, carbon monoxide, nitrogen dioxide, and volatile organic gases. A number of studies have demonstrated this fact for specific pollutants:

- Ott (1971) collected CO samples in San Jose, California at breathing height within a 33 square kilometer grid. The official air monitoring station, located near a street with heavy traffic, recorded concentrations approximately 100 percent higher than the background level. Samples were also collected by pedestrians walking along congested streets. These values were on average 60 percent higher than the air monitoring station values.

- Cortese (1976) obtained similar results for commuter exposure to CO in Boston. The average of the maximum 1-hour personal exposure concentration (10.8 ppm) was approximately 1.4 times the corresponding average concentration (7.8 ppm) measured at the two metropolitan air monitoring stations. About 10 to 15 percent of the difference could be attributed to the height of the fixed station sensors (15 feet) which were about 10 feet above breathing heights. CO levels are known to decrease as height increases.

- EPA's Office of Pesticides and Toxic Substances' 5-City Study compared personal monitor and fixed station measurements for eleven volatile organics. In every case, the fixed station badly understated the exposures of the subjects. For example, applied to a city of a million inhabitants, the fixed station results would indicate that 400,000 people were not exposed to chloroform above the detectable limit, while the personnel air samples show the true number is more like 50,000 - an eightfold overestimate of the unexposed population by the fixed stations.

These results bring into question the accuracy of population exposure levels based on ambient levels. One recent study of exposure to respirable particulates and sulfates however did find that over a large range of outdoor concentrations measured, mean exposure levels as measured by personal monitors could be correlated with mean outdoor levels (Dockery and Spengler, 1981). This may imply that measurement of ambient pollutant levels can be useful for certain pollutants in certain cities and may be related in a simple way to population exposures under certain conditions.

There have been a number of attempts to develop refined approaches to exposure assessments based on ambient data. For example, an approach has been developed (EPA, 1976) which merges demographic data with air quality data. The approach assumed individuals spent 24 hours per day at their residences. A grid of 215 points in the New York - New Jersey - Connecticut area was formed and the concentration at each point in the grid estimated by interpolating levels measured at the three nearest air monitoring stations to each grid.

One problem with this approach is that the number of valid sampling sites is too small to give sufficiently precise spatial resolution of concentrations, thus requiring extensive extrapolation from limited data. This is particularly troublesome when one considers that the New York - New Jersey - Connecticut area has more sampling sites than many other areas. There are two other major problems with the approach that are also at the heart of the problem involved in using ambient concentration data to estimate population exposure levels:

- People do not spend 24 hours per day in the same place, but rather move during a day to areas of higher and/or lower pollutant concentrations while engaged in recreation, occupational, or travel activities.

- Sampling stations are usually located outdoors, but people spend as much as 90 percent of their time indoors where exposure to a particular pollutant might be higher or lower than outdoor levels.

To accurately determine actual exposures, it would be necessary to possess data on concentrations at every point (indoors and outdoors) that a person is present throughout the day, not just observations from an outdoor sampler. Various studies (Repace and Lowrey (1980), for example) have found great differences between indoor and outdoor levels of pollutants. Sources for some pollutants such as CO and nitrogen oxides can be generated indoors and consequently indoor levels of the pollutant can even be higher than outdoor levels.

### 3.2.2 Use of Dispersion Models in Estimating Exposure Levels

A common approach to the estimation of exposure levels to a pollutant is the use of environmental transport and transformation models to predict concentration levels in the various media on the basis of the specific characteristics of the pollutant. The most common mathematical models of the dispersion of airborne pollutants from point sources are based on gaussian dispersion characteristics. Equations are developed to estimate concentration of a pollutant at a point. Parameters of an air dispersion model might include the following (Eifler, *et al*., 1981):

- number of wind direction sectors;
- emission rate;
- wind speed;
- index for Pasquill stability class;

- index referring to the proper wind direction sector in which the line from the receptor to the point source lies;
- meteorological joint frequency function of wind direction sector, wind speed category, and stability class;
- height of the point source;
- wind speed at height of the point source;
- distance from the point source to the receptor point;
- vertical standard deviation of the pollutant concentration as a function of downwind distance and of stability class; and
- effective height of the point source.

The concentration at a point is obtained by summing across the contributions from all point sources. The model can also be used to develop isopleths of concentrations.

It can be seen that air dispersion models, especially for a number of sources, can be very complex. In fact, it has been observed that most air pollution dispersion models are unsuited for exposure assessment, being too complex to operate, too data-intensive, or too site-specific (Dennis, 1980). Under the right conditions, however, air dispersion models can be an effective approach to estimation of exposure levels, and for prospective analysis, few alternatives may be available.

Previously we mentioned that two major problems associated with use of ambient concentration data to estimate population exposures are that people are mobile and spend a considerable amount of their time indoors (while ambient data relate to outdoor levels). For air dispersion models, matching the exposure levels with the location of people as they move within and out of the geographical area involved remains a problem. The indoor-outdoor situation may however be able to be addressed in the model. Moschandreas and Stark (1978), for example, developed a model to predict the indoor air pollution levels by simulating a series of complex interactions involving outdoor pollutant levels, structural characteristics of the examined buildings, and behavioral patterns of the inhabitants. The model was validated with a large set of observed data. It was able to predict indoor pollution concentrations within 20 percent of the observed hourly indoor values for CO, NO, $NO_2$, $CO_2$, and nonmethane hydrocarbons.

In some situations dispersion models cannot be developed and applied. For example, Nagda (1979) reported that a refined air quality model for trichloroethylene sources could not be developed because data on stack parameters were unavailable and there was uncertainty in the atmospheric reactivity of trichloroethylene. In this case, the available emissions data were used to compute emission densities and high/low exposure areas.

Mathematical models can also be developed to predict concentrations of pollutants in drinking water and food on the basis of the specific characteristics of the pollutant and of the environment thorough which it passes. For example, a principal Water Treatment Processes (WTP) model has been developed for use in predicting the degree of removal of a toxic substance (Hedden, 1980). When linked with an aquatic fate and transport model, a source identification and loading model, and a model for estimating the population at risk, the WTP model can be used to predict the amount of a toxicant actually reaching a population for consumption by the drinking water route.

In summary, dispersion models represent a viable alternative approach to the use of ambient concentration data for purposes of population exposure estimation. However, they can become quite complex and difficult to use and apply to exposure assessment. They also share with the exposure assessment techniques based on ambient data the problem of relating the outdoor exposure levels to the actual exposures received by people within the area. Their major advantage is that they can be used to prospectively analyze the effects of changes in pollutant emissions air exposures. In section 3.2.4 we discuss alternative methods that are designed to address the weaknesses of the previously described approaches. We first discuss in section 3.2.3 the estimation of population-at-risk.

### 3.2.3 Population-at-Risk Estimates

In addition to estimates of the concentrations to which people are exposed, an exposure assessment must include estimates of the numbers of people exposed to those concentrations. Most studies rely on Census data of residential living patterns (Miller, 1978). Census data reveals numbers and characteristics of people and their dwelling units. Census data are reported by geographic units (Census tracts) which typically have about 4,000 people. Tracts are relatively small in size near densely populated areas and progressively larger as one goes toward suburban and rural areas.

Exposure assessments commonly assume that dwelling units are evenly distributed throughout the area under study or throughout individual tracts. This assumption introduces error into the exposure assessment by the degree to which the actual population distribution varies from the assumption and by the degree to which exposure levels within the area (e.g., Census tract) vary.

Significant errors can potentially be introduced to an exposure assessment by this assumption. Rydell, et al., (1968) found that redistributing population from high density central zones to low density fringe zones would accomplish a 30% reduction in air pollution damage from urban sources.

Other approaches have been utilized to estimate populations-at-risk. For example, one study involved use of an earth satellite to grossly discern population patterns within a city (Todd, et al., 1979). This information was used with Census data for the city to develop estimates of population densities within segments of Census tracts.

One major problem with all these approaches is that they assume that people spend all their time at their dwellings. In fact, people spend part of their time away from home. Error is introduced into the analysis to the extent that there is a difference between exposure levels at dwelling units and away from dwelling units. For pollutants with no threshold and linear dose-response relationships and within cities where movement of people within the city does not reflect a strong pattern, the error introduced into the risk assessment could be small. However, in general there is sufficient nonhomogeneity in population densities even within relatively small areas to result in the introduction of a sizable degree of error in exposure estimates made through the use of these procedures.

In recent years, an alternative approach has emerged for estimating the numbers of people exposed to various concentration levels. This approach does not assume that people spend all their time at their dwelling units. It involves defining "microenvironments" which are geographical or locational settings within which exposure levels are suficiently uniform to allow simple assessments of exposure on the basis of activity or time use patterns. One set of microenvironments used to characterize exposure are those used by the Ambient Standards Branch of EPA's Office of Air Quality Planning and Standards (EPA, 1981e):

- Indoor work
- Other indoors
- Inside transportation
- Other transportation along roads
- Other outdoors

Time-budget data from the Nationwide Personal Transportation Study (NPTS) of the U.S. Census Bureau were used to distribute people among these five microenvironments. In this way estimates are developed for the number of people exposed to the concentration levels associated with the microenvironments.

As the specification and characterization of microenvironments improves, time-budget data must also be improved. Not only is it necessary to obtain accurate data on the duration that people spend in selected microenvironments, data are also required on the successive combinations of microenvironments that people occupy throughout the day (EPA, 1981e). Existing activity pattern studies have not obtained these necessary duration and transition data for the microenvironments currently being constructed for pollutant exposure.

EPA has designed a research project which has as one of its goals the development of detailed activity pattern data for a city (EPA 1981e). The study design prescribes the use of both random and stratified samples of the population to ensure that the results are representative of the general population. This study plan also integrates the definition of various microenvironments into the definition of possible daily activities. This study, when implemented, may provide the first detailed documentation of time budgets which can be easily translated into the microenvironment framework. To develop population exposure estimates with confidence, the study would have to be repeated for a variety of different urban and non-urban area types. Representative activity patterns for major classes of location (e.g., southwest urban, southwest non-urban, northeast urban, etc.) may emerge from these analyses. These activity patterns could then be used to develop population exposures based on microenvironment exposure concentrations.

This section has reviewed the methods used to estimate the population-at-risk to an environmental pollutant. Methods in this regard associated with the use of microenvironments were briefly discussed. In the next section, we discuss in more detail the use of microenvironments as well as the use of personal monitors for purposes of exposure assessment.

### 3.2.4 Recently Developed Exposure Assessment Methods

The majority of past exposure assessments have estimated potential exposures rather than measured actual exposures. These assessments have typically assumed that people remain in one place and that exposure levels are constant over a geographic area that includes both indoor and outdoor areas.

The theoretically correct approach to exposure assessment involves expressing exposure as a function of an individual's place in time and space in relation to the spatial and time coordinates of the concentrations of a pollutant (Ott, 1980). With this type of information for all individuals in an area, a frequency distribution of exposures could be developed. This type of information could be used to assess the population's risk from the pollutant. In this regard, frequency distributions could be developed for each population subgroup of concern (the aged, infirm, special occupational groups, persons especially susceptible to the pollutant of interest).

Two alternative approaches have emerged that attempt to improve measurement of exposures of the population to air pollution:

- Field measurement of the exposures of a representative population using personal exposure monitor (PEM) instruments.
- Development of data on activity patterns of the population and the pollutant concentrations expected in microenvironments.

The "ideal" exposure assessment, irrespective of resource constraints, could involve equipping everyone in a city with a PEM instrument for a long period of time. Population exposure could then be described fully and with high accuracy. This approach would, however, be very costly and logistically unworkable.

A more practical alternative approach would be to use a representative sample. In an urban area of several million persons, only a relatively small sample (less than a thousand persons) would be necessary to estimate exposures with statistical precision for the entire city. Of course, the sample would need to be carefully selected to properly represent various subpopulations such as the aged or persons especially susceptible to the pollutant being studied. Developing such a sample and gaining individuals' agreement to participate in the study could be a major source of difficulty.

This method of exposure assessment is dependent on the availability of PEM instruments for the pollutants of interest. The instruments must be small, rugged, and capable of operating for long periods on small, light batteries. As discussed in Chapter 2, such instruments are currently available for only a very limited number of pollutants.

An exposure assessment based on personal monitoring data from a representative sample of the population can produce a good description of the population's current exposure situation. The approach cannot be used to predict how the current exposure might change as pollution sources change. For policy setting purposes, exposures over time must also be able to be predicted. Predictive capability depends on establishment of cause-effect links, which requires a computational scheme or model of some kind. Such a scheme can be represented by the microenvironmental exposure assessment approach mentioned previously.

In Chapter 2 we described the concept of microenvironments in regards to assessing an individual's exposure to an environmental pollutant. Population estimates can be developed by combining data on the times that people spend in selected microenvironments with estimates of the concentrations likely to be experienced in these microenvironments.

In conducting a population exposure assessment by use of an approach involving microenvironments, one of the most important tasks is to identify all the appropriate microenvironments. The activities and movements of people as a function of time during the day must also be adequately represented. Because unique exposures often occur in very small physical locations and for relatively short periods of time, (passenger compartments of motor vehicles, rooms in houses, etc.) it is necessary to treat the spatial and temporal properties of air pollution with a very fine resolution and in considerable detail. The theoretical criteria for selecting microenvironments needs further development.

Estimation of the average exposure of a population is computationally straightforward. Estimation of the frequency distribution of exposures is more difficult computationally, but computer simulation and other techniques can be used. For example, SHAPE (Simulation of Human Air Pollution Exposures) uses simulation techniques to combine data on activity patterns of the population with statistical descriptions of pollutant concentrations in specified microenvironments (Ott, 1981). The SHAPE model computes the average exposure, standardized exposure, moving average standardized exposure, maximum moving average standard exposure, and maximum hourly exposure. Computations are repeated for a large number of persons to generate the frequency distribution of exposure.

Exposure estimates from SHAPE currently have limited applicability because the activity pattern data used are very limited and somewhat outdated, and the concentration data on which the microenvironmental subroutines were based often are very limited and not necessarily representative (EPA, 1981c). As the model is currently implemented, for example, the concentration of pollutants within microenvironments does not vary over the year.

Pollutant concentration data for exposure assessments involving microenvironments can be developed from PEM samples, ambient fixed station monitoring networks, or dispersion models. If monitoring network data are used, values for specific microenvironments must be derived by modeling or interpolation techniques. For CO, only a relatively few activities have been associated with important CO exposure levels (EPA, 1981e). In all other activities, it appears that CO exposures can be estimated accurately using concentration data obtained from fixed station background monitors, and it is likely this finding may apply to other air pollutants.

The microenvironmental approach to exposure assessment can be used to predict future exposures by changing input parameters consistent with different regulatory scenarios. This is one advantage over the personal monitor approach to exposure assessment. It should be pointed out however that the microenvironmental approach needs to be validated with actual exposure data and an accurate assessment of total exposures will require the balanced application of both techniques.

In summary, a number of factors need to be considered in selecting an exposure assessment approach. The requirements of the risk assessment regarding exposure data must be considered in developing the exposure assessment. The availability of dispersion models, PEM instruments, and other resources must also be considered. Finally, the exercise of professional competence has been identified as the most important ingredient in exposure assessment at the present state of development of this field (EPA, 1981c).

In the next section, we review the issues involved in and past efforts directed at measuring the population's total exposure to a pollutant or group of pollutants from multiple sources or medias. Section 3.2 has mostly focused on the air medium. The next section will consider all of the main routes for human exposure -- air, water, and food.

## 3.3 ASSESSMENT OF TOTAL POPULATION EXPOSURES

### 3.3.1 Introduction

Exposure assessments often focus on one pollutant in one environmental medium. Most pollutants however are not confined to a single medium. In fact, pollutants entering the environment through one environmental medium can pass to other media one or more times. Examples of intermedia transfer are:

- The wet and dry removal of pollutants from the air,
- The resuspension of soil-deposited pollutants in the air,

- Vaporization of pollutants from soil and water, and,
- The leaching of pollutants from waste disposal piles by groundwater.

One reason for the focus on single media relates to fragmented agency responsibilities. Even within agencies, because of the organizational arrangement, studies may be devoted exclusively to one medium although major contributions to exposure may be through other media. This may be due to tradition, past experience of individuals in the program, or narrow interpretations of missions. Pollutant transport within a single medium is often much easier to model than transfers from one medium to another.

Often, people are faced with exposure to multiple pollutants from air, water, and food. Exposure assessments focusing on a particular group of people or a particular pollutant need to consider human exposure through all media. Recently, recognition of this fact has resulted in efforts being directed at developing techniques and actually assessing total human exposures to environmental pollutants.

A prime objective of total exposure assessments is to identify the most important pathways of exposure for each compound and the most likely biological target for sampling. This allows for appropriate control of a pollutant for segments of the population most adversely affected by exposure to the pollutant. With good information on the relative importance of the various pathways for a particular pollutant, it may be possible to eliminate some pathways as of very minor concern. However, simplification to a single pathway through one medium will rarely occur in the ambient environment (Behar, et al., 1979).

In section 3.3.2 we briefly discuss several total exposure conceptual models. Then in section 3.3.3 we discuss recent total exposure assessments. Several of these were conducted to further develop a methodology for total exposure assessment, while others were conducted as part of a risk assessment.

### 3.3.2 Total Exposure Models

Behar, et al., (1979) developed the Integrated Exposure Assessment Monitoring (IEAM) concept. It involves the coordination of environmental (air, water, land, and crops) monitoring networks to systematically collect pollutant exposure data for a specific receptor - usually man. The IEAM approach provides data required for strategic control of critical sources of pollutants that cause major problems or threats to critical receptors which are defined as follows:

- Critical receptor - Segment of the human population or of the environment most adversely affected by exposure to a pollutant.
- Critical sources - Major contributors to exposure of the critical receptors.

Because of scarce resources, it is important to assure that monitoring measurements are directly related to the critical sources.

Behar stresses the need to carefully consider all sources for a pollutant. For example, particulates settle out of the air on surfaces and are then resuspended by the motion of cars, people, etc. Ambient air concentrations may increase exponentially as the surface is approached. Receptors such as small children, whose breathing zones are close to these surfaces, breathe air which may contain 3-10 times the particulate concentrations thought to be experienced by an adult. Realization of the overriding importance of this secondary source is a recent development. Ingestion of surface particulates may also occur for children. In situations such as this, Behar points out the need for short-term special studies. These would consider breathing heights of children, resuspension factors, and ingestion habits of children. Other factors which affect the relationships between pollutant exposures and dose levels in children are discussed in Chapter 4.

Drake (1980) addresses the issues involved in interfacing air pathway models with other media models to allow consideration of intermedia transfers of pollutants. In order to model the fate of pollutants along the pathways, the investigator must know:

- how these intermedia transfers occur,
- time-lags induced by the transfers, and
- nature of the important processes in each medium.

Drake focuses on these intermedia transfers. He presents a compartment model which treats the passage of pollutants through the pathways as a set of interacting compartments with transfer functions linking one compartment to another. Compartments may represent the air medium, a lake, soil, a plant or animal species, or a given organ of the human body. Equations are developed for the rate of change of pollutant concentration in each compartment.

Walsh, et al., (1978) developed a hazard assessment methodology for limiting human exposures to environmental pollutants from multimedia sources such that exposure or dose will not exceed some preselected value. Mathematical formulations which relate hazard measures to environmental pollutant concentrations are developed for each sampling medium corresponding to each effluent mode. Simultaneous exposure to several pollutants may result in synergistic or antagonistic effects rather then simple additive effects. The model can be applied to multiple pollutants only if the multiple pollutants affect the same target organ and influence the same biological effects. Research may eventually lead to a comprehensive approach for determination of composite hazard measures. This model is discussed in more detail in Chapter 4.

These conceptual models provide a framework from which to view the assessment of total population exposure to a pollutant or group of pollutants. In the next section, we review several exposure assessments which have considered multimedia sources.

### 3.3.3 Total Exposure Applications

Within EPA, several programs appear to routinely consider multimedia pathways when conducting an exposure assessment. The Office of Pesticide Programs has developed methods to consider actual dermal, inhalation, and oral exposures to pesticides. Oral exposures occur through food consumption. Tables of food consumption and data on the extent to which crops are currently treated with the pesticide of interest are used. Exposure estimates for the three routes of exposure are combined to produce the total exposure assessment (EPA, 1981c).

The Office of Radiation Programs also considers multimedia sources in their exposure assessments. Solid, liquid, and gaseous radioactive effluents containing many radionuclides are emitted from nuclear reactors and expose populations through pathways of inhalation, ingestion, and direct radiation. Data (reactor emissions, meteorological, etc.) are used to estimate radionuclide concentrations in air; groundwater; surface water; and in such foods as meat, milk, and fresh produce. Ingestion doses are based on the assumption of uniform daily consumption rates for each type of food.

The Office of Water Regulation and Standards' exposure assessments are limited to exposure via water media. However, an attempt is made to put all routes of exposure into perspective. The Office of Toxic Substances also considers routes of exposure through all media in exposure assessments it performs.

EPA's Office of Research and Development has been active in development of multimedia exposure assessment techniques. Several important research studies in this regard have recently been conducted. We first discuss two of these studies and then discuss two exposure assessments for particular pollutants which considered multimedia sources.

EPA conducted a pilot study in Beaumont, Texas and Chapel Hill, North Carolina to field-test methods for measuring human exposure to a number of volatile organic compounds (VOC) and to compare levels of VOC in breathing-zone air and drinking water with levels of the same compounds in human breath (Zeidinger, et al., 1981). Beaumont was selected to represent a petrochemical manufacturing area while Chapel Hill was selected to represent a non-industrial area.

Students in each city carried air monitors and water sample vials for use each time they drank water. Breath samples were taken at the end of each day. The students were administered a questionnaire to determine factors possibly related to exposure, such as residence on or off campus, dietary habits, parents' occupations, etc.

The study found great variability in exposures for seven of the 10 most prevalent compounds. This indicates the errors that would have been associated with characterizing either student group as a geographical cohort with uniform exposures. This could have important implications for epidemiological studies, which have traditionally assigned similar exposure histories to residents of a given region. These findings could not have been made using standard approaches of ambient monitoring. Preliminary results of a similar study in individuals in New Jersey have also been reported (Wallace and Pellízzari, 1982).

The most ambitious total exposure assessment research project in recent years is the Total Exposure Assessment Methodology (TEAM) study with the goal of field testing methodology for estimating total human exposure to selected hazardous substances (Pellizzari, et al., 1981). Phase I of TEAM was a pilot study involving 9-persons and was completed in December 1980. Its purpose was to field-test about 30 candidate sampling and analytical methods for measuring environmental and biological concentrations of four classes of compounds: volatile organics, metals, pesticides, and polyaromatic hydrocarbons.

The pilot study found a number of volatile organics to vary greatly (3-4 orders of magnitude) in air and breath concentrations from one individual to another. This indicates that sources are likely to be small, localized, and intense, rather than varying smoothly over some defined geographic region (EPA 1981e).

Phase II of TEAM has been initiated. It involves monitoring 375 New Jersey residents and 25 Greensboro, North Carolina residents for exposure to 22 volatile organics in their air and drinking water. Concurrently, the same chemicals or their metabolites are measured in each person's exhaled breath. The study attempts to establish for each chemical the relative importance of certain routes of exposure and whether a predictable correlation exists between exposure and body burden.

Recently, a powerful tool for characterizing population exposures to pollutants has been developed by the EPA Office of Pesticides and Toxic Substances. The Graphical Exposure Modeling System (GEMS) incorporates a large number of models for characterizing the environmental dispersion of pollutants in air, surface, and groundwater with data bases involving population distribution, activity patterns, and the geographical distribution of potential and actual sources of pollutant emissions for the entire United States. Specifically, the system includes (EPA, 1982) the following models:

- ATM/SECPOP, which integrates atmospheric dispersion estimates from point sources of air pollution with population density data to develop estimates of population exposure to air pollutants;
- PTMAX, which describes short-term concentration of airborne pollutants generated by a single point source;
- BOXMOD, which describes the dispersion of air pollutants from diffuse area sources, such as metropolitan areas;
- EXAMS, which traces the diffusion of pollutants in aquatic ecosystems;
- SESOIL, which describes the movement of pollutants through the unsaturated zone in soil;
- AT-123D, which models the movement of pollutants in groundwater in the saturated zone, and
- ENPART, which assesses the distribution of chemical pollutants between various air, water, and soil compartments.

In addition, GEMS contains several models for assessing the physical properties of pollutants related to environmental fate and transport, based on their chemical structure. These properties include volatility; partition coefficients between air, water, and soil; stability in air and water; and propensity to be bioconcentrated.

The GEMS system is supported by a number of data bases including:

- 1980 Census data accessible at the Census tract level, by zip code, or by latitude and longitude specification;
- a file which describes the amount of time spent in one of five defined microenvironments (indoors, work/school; indoors, other; inside transportation vehicle; other transportation; other outdoor) by individual in four different SMSA's in twelve age/occupational categories;
- climatic and meteorological data for the entire United States; and
- 1977 economic Census data which describes the geographic distribution of industrial facilities by SIC code.

The purpose of the GEMS system is to allow the assessment of population exposures to pollutants given a defined pattern of emissions. Potential applications include the assessment of population exposure resulting from air pollution emissions and from hazardous waste facilities, and the identification of important routes of exposure for multimedia pollutants.

Currently, only some of the models and data base have been fully integrated into the GEMS system. That is, not all relevant data bases can be used directly as inputs to all models and not all models can be linked together to provide exposure assessments (EPA, 1982).

## 3.4 RESEARCH NEEDS

A key component of a risk assessment of an environmental pollutant is the assessment of the population's exposure to the pollutant. Methods commonly used for population exposure assessment have been demonstrated to yield results with significant levels of error. New population exposure assessment approaches, such as those based on the study of microenvironments, appear to offer the opportunity for significantly improved exposure assessments.

It appears to us that with the importance of the results of population exposure assessments, the promise of the new approaches, and the problems with the commonly used approaches, this area clearly is one that should receive high priority research attention. It also seems to us that those in the EPA Office of Research and Development currently working in this area are in the best position to specify the details of needed research. Based on our review, however, we believe that research within the following three general areas is needed:

- Development of an EPA strategy for population exposure assessment,
- Development of better data needed for exposure assessments, and
- Development of improved exposure assessment study designs.

Each of these are briefly discussed below.

Different programs within EPA have different ideas as to appropriate exposure assessment techniques. Part of this, no doubt, is based in tradition and past experience of the individuals in the different programs. Some of the difference, however, is based on different requirements for the exposure assessment. For example, average exposures may suffice for some purposes and exposure assessments based on ambient concentration data may also suffice for this purpose for a particular pollutant. There seems to be a need for an EPA exposure assessment strategy that helps identify the appropriate approach to follow in different situations. The strategy could also address issues such as the frequency that population studies might need to be repeated to take account of changes in exposure and population parameters.

The second research area deals with the development of better data for exposure assessments. Many previous exposure assessments have relied heavily on assumptions regarding such things as human consumption rates for different foods. More precise data in these situations would improve the accuracy of the exposure assessments. There is also a need, on occasion, for special studies for specific exposure situations, e.g., amount of lead exposure faced by children because of hand-to-mouth activity. One additional research need in this category regards the need to develop better baseline exposure data to compare results of monitoring "unhealthy" areas.

The third research area involves the development and implementation of improved exposure assessment study designs. The microenvironmental approach to exposure assessment, while very promising, has not been fully developed and validated. Among the specific areas requiring research attention are the following:

- refinement of approaches to selection of population samples,
- development of theoretical criteria for selection of microenvironments,
- analysis of intercity variations in activity patterns and definition of microenvironments,

- characterization of pollutant concentrations in microenvironments and variability over time,
- development and validation of predictive exposure assessment models, and
- analysis of alternatives for dealing with subjects regarding recording exposure dates, food intake, and activities during the day.

In summary, there is a clear need for improved data on the population's exposure to environmental pollutants. Techniques for production of improved data are in an early stage of development. We believe that research focused on the further development of these techniques and on related areas, is clearly needed and can have an important impact on improved environmental protection.

# 4. RELATIONSHIP BETWEEN PATTERNS OF EXPOSURE AND DOSE OF POLLUTANTS

## 4.1 INTRODUCTION

A number of important issues arise when analyzing the relationship between observed patterns of human exposure to pollutants and their relationships to the magnitude of physiological and health impacts that occur as a result of those exposures. One of the basic principles of modern toxicology is that the magnitude of the effect associated with any exposure to a toxic substance is related to the amount of the substance that reaches the target organ (Klaasen, 1980). In the case of environmental exposure to pollutants, there is often a complex relationships between the amount of substance present in the vicinity of the exposed individuals and the amount of the substance that reaches the target organs. This complexity is a result of wide variations in the degree to which pollutants are taken into the body; absorbed into the blood stream; and transported, metabolized, stored and eliminated from the body.

This chapter deals primarily with three main aspects of this problem. The major focus is on recent progress that has been made in elucidating the relationships between exposure patterns and dose levels, that is, factors which govern the extent to which pollutants are absorbed by the body. Both the properties of pollutants and exposed groups which affect these relationships are discussed.

The second aspect discussed is the rapidly-advancing research in the evaluation of physiological indicators of exposure and dose other than direct toxic effects. Recent studies are reviewed and the current state of knowledge about these indicators, e.g., the significance of body burden or blood metabolite levels as a measure of exposure and dose, is discussed.

The final aspect discussed is the mathematical and statistical models that have been developed for the analysis of exposure-dose relationships for environmental pollutants. Classical pharmacokinetic models for some pollutants are briefly discussed, but the major focus is on recently-developed models for the comparison of the relative contribution to total dose of various routes of exposures and for the comparison of the magnitudes of exposure and dose of pollutants from different sources in the environment. The voluminous literature on the metabolism of environmental toxicants in the body, which is far beyond the scope of this chapter, is dealt with only when it is directly relevant to a major topic of discussion.

The chapter concludes with a brief outline of what appear to be important gaps in knowledge about the relationship between exposure and dose levels for environmental pollutants and possible areas for future research.

## 4.2 RELATIONSHIPS BETWEEN EXPOSURE AND DOSE

### 4.2.1 Properties of Pollutants Affecting Exposure-Dose Relationships

From the point of view of environmental risk analysis, the issue of how the properties of pollutants affect dose levels arises primarily in the case of particulate pollutants where particle size and solubility have long been known to be important determinants of exposure-dose relationships. A large part of the following discussion thus deals with recent studies of factors which effect the uptake of particulate pollutants through the lung and alimentary tract. The absorption of gaseous pollutants is less often an issue in environmental exposure-dose analysis because the uptake of these pollutants through the lung during the characteristic low-level exposures is thought to be essentially an equilibrium or steady-state phenomenom governed primarily by the solubility of the substance in question. We address this topic only briefly, placing the emphasis on the absorption of organic pollutants through the alimentary tract where the relationships are more complex and relevant to the environmental setting. Dermal exposure is not discussed because, in the environmental setting, this route of exposure is relatively unimportant as a determinant of total exposure.

The principal physical properties of particulate pollutants which govern the extent to which they are absorbed are the particle size distribution, shape and solubility (Menzel and McClellan, 1980). The basic principles governing the extent to which particulate matter is deposited in the lung have long been understood, and recent advances in this area have mostly been refinements and additions to the standard models for lung deposition such as that developed in 1966 by the International Commission on Radiation Protection (ICRP, 1966). Attempts have been made to refine this model to take into account individual or population variation in breathing patterns and the properties of individual pollutants. The basic principles of the ICRP model have survived with relatively few revisions (Camner, _et al_., 1979), and provide the basis for many recent attempts to model relationships between particulate exposure and dose (Mendez and Hattis, 1978; Gasper and Dausvardis, 1979; MITRE 1979, 1979a; Medinsky, _et al_., 1981).

Because particles with appreciable solubility deposited anywhere in the respiratory tract (not just in the lung) will ultimately either be absorbed through the alveoli walls, solubilized in the lymph nodes, or partially or completely absorbed in the alimentary tract, knowledge of the initial site of particle deposition, while important, is not suffient to allow accurate prediction of the extent of uptake that will occur after exposure to a given pollutant. Knowledge of the routes and rates of absorption of the pollutant is also required. A number of recent studies of particulate pollutants have addressed the issue of absorption and retention of particulates after inhalation, primarily for metallic aerosols.

Perhaps the best-studied metallic air pollutant is lead. The earliest studies of lead particulate absorption through inhalation found that adult male subjects exposed to very fine particles of lead sequioxide absorbed about 30-50% of the inhaled particles (Kehoe, 1969). This corresponds to more or less complete absorption of the entire deposited dose of this relatively soluble salt. More recent studies, both epidemiological and in the laboratory, have suggested that this is typical of absorption efficiencies observed in adults exposed to lead in urban air . Absorption efficiencies observed in different populations, exposure conditions and lead compounds range between 20 and 50 percent absorption within a relatively short period of time (days to weeks) (MITRE, 1979; Morrow, et al., 1980). Less efficient absorption of less soluble salts of lead has been observed (NAS, 1972; OSHA, 1978) but the bulk of the particulate lead found in the ambient urban environment seems to be composed of the relatively more soluble salts (EPA, 1977).

Lead particles larger than about one micron will be deposited most frequency in the nasopharynx and trachebronchial region, where they will be rapidly transported by ciliary action to the pharynx and swallowed. Studies of exposure to larger diameter particles (less typical of environmental exposures) suggest that such indirectly ingested particles are absorbed with an efficiency of about 8-10% (NAS, 1980). Because particle size distribution may be highly heterogeneous, with a relatively large fraction of total mass in a few large particles, it is clearly crucial when estimating the dose of lead received by any exposed population to know the size distribution of the particulate involved. Recent research suggests that, on the whole for a given exposure level, larger particle size implies generally lower absorption efficiencies (Exhibit 4-1). This would apply to all particulates, not just lead.

As for indirectly ingested lead particulate, lead absorption from dietary sources through the alimentary tract is thought to be relatively inefficient, with approximately 8-16% being absorbed in nonfasting, healthy adults (NAS, 1980). Fasting adults and normal children appear to be much more efficient at absorbing dietary lead, with as much as 53 percent of the administered dose being taken up (MITRE, 1979).

More or less the same principles which govern the uptake of lead from the respiratory and alimentary tracts apply to the uptake of other metallic particulate pollutants. Ranges of absorption for other well-studied metallic pollutants are summarized in Exhibit 4-2. Most of the metals are relatively poorly absorbed in the gastrointestinal tract, with the exception of mercury, whose uptake is facilitated by the relatively high lipid solubility of many of its compounds. Uptake through the respiratory tract (in this case, the sum of

## EXHIBIT 4-1

## CALCULATED TOTAL ABSORPTION OF METAL PARTICULATES AS A FUNCTION OF PARTICLE SIZE a/

| Particle Size (MMAD) (microns) | Percent Absorption When Alveolar Absorption is: | |
|---|---|---|
| | 50% | 100% |
| 0.1 | 50.4 | 26.7 |
| 0.5 | 30.8 | 16.6 |
| 2.0 | 22.2 | 12.6 |
| 5.0 | 13.4 | 8.6 |
| 10.0 | 9.2 | 6.8 |

---

a/Assumes gastrointestinal absorption efficiency = 5%.

b/MMAD = mass-median aerodynamic diameter

Source: Camner, 1979.

## EXHIBIT 4-2

## ESTIMATED ABSORPTION EFFICIENCIES FOR METAL PARTICULATES IN THE GASTROINTESTINAL AND RESPIRATORY TRACTS

| Element | Absorption (%) | |
|---|---|---|
| | Gastrointestinal Tract | Respiratory Tract |
| As | 1-5 | 50-55 |
| Cd | 3-10 | 10-40 |
| Cr | 5-25 | 10-25 |
| Hg | 25-95 | 65-90 |
| Pb | 5-10 | 20-37 |

Source: Gasper and Dauzvardis, 1979.

material absorbed in the lung and indirectly ingested after inhalation) is somewhat more efficient. The primary reason again is the efficient absorption solubilization of small particles in the alveoli.

The figures given in Exhibit 4-2 are the total absorption of particulate calculated after an indefinitely long period of time. There is considerable variability in the speed with which clearance from the lung by one mechanism or another actually occurs, with the small soluble particles absorbed in the alveoli rather rapidly (less than one day), small insoluble particles transported slowly (weeks to months) to the thoracic lymph nodes, and large particles being eliminated (if not dissolved) through the alimentary tract in several days.

The only organic particulate matter of interest to environmental policy is probably soot in one or another of its forms: the product of coal combustion, coke oven operations, or the combustion of diesel fuel. Few generalizations can be made about the properties of this class of pollutant, other than that the particle size encountered in the environmental setting is generally quite small so that alveolar deposition is likely. The elucidation of exposure-dose relationships is complicated by the fact that these pollutants tend to display great heterogeneity in chemical composition and tend to absorb and transport organic and inorganic pollutants into the alveoli.

The uptake of organic chemical vapors and gases through the lung, as described earlier, takes place primarily by diffusion. It is driven by the difference in the partial pressures of the inhaled substance in the alveolar air in the blood (Klaasen, 1980, Fiserova-Bergerova, 1981). The rate of uptake is proportional to this concentration difference and the solubility of the gas in lipids and water, with lipid-soluble vapors being transported faster than lipid-insoluble substances. The rate of uptake and the total absorbed dose also depend on the relative affinities of the substances for the various organs in the body and the rate at which the substance is metabolized, with more rapidly-metabolized substances being absorbed more rapidly due to the more effective maintenance of blood-air concentration gradients.

Experimental studies and modeling of the time course for absorption have indicated that for a number of simple organic vapors and gases, equilibrium between the concentrations of vapor in the inhaled air, body tissues, and exhaled air is established rather rapidly (on the order of a few hours at high concentration). This result should hold at exposure levels commonly encountered in the environment, because most of the steps in the process should be first-order at low concentrations (Fiserova-Bergerova, 1974, 1981). A number of studies have made use of this principle and explored the use of measurements of volatile organic solvents in exhaled air to provide indices of environmental exposure (Verberk and Scheffers, 1980; Wallace, 1983). These studies are discussed in more detail later.

For unmetabolized substances, the net "dose" is zero, because all of the substance absorbed during an exposure will ultimately be exhaled or excreted by other routes. For a substance which is metabolized, the "dose" of substance (the amount of substance which is not eliminated at the end of exposure) is equal to the amount metabolized. This concept of "dose" is of course deceptive. Even substances which are not rapidly metabolized are present in significant concentrations in the blood and organs during exposure and it may well be that some of the so-called "nonmetabolized" substances do in fact interact physically or chemically with target organs to produce damage. In addition, there may be multiple metabolic pathways with only one or a few of them actually producing damage. Thus, when analyzing environmental exposures to organic vapors and gases, it is customary to estimate "dose" in term of integrated exposure, occasionally modified by some simple approximation for uptake based on solubility considerations (Carcinogen Assessment Group, 1980).

As far as gastrointestinal absorption of organic substances is concerned, the major areas of interest in environmental risk assessment and exposure analysis are, 1) halogenated solvents and other low molecular-weight hydrocarbons, and 2) persistent pesticide residues in food and water. The conventional wisdom regarding these substances is that the efficiency of gastrointestinal uptake will be proportional to the lipid-solubility of the compounds in question (Klaasen, 1980), but in practice, the situation is by no means so simple and the nature of exposure-dose relationships is not well understood.

For low molecular-weight organic hydrocarbon impurities in drinking water, little is known about the extent of uptake through the gastrointestinal tract at low concentration. Risk assessment has generally proceeded on the assumption that absorption is quantitative, or that, given the scarcity of data, assessment of risk based an integrated exposure, rather than dose, is appropriate (NAS, 1977). The assumption of essentially quantitative absorption for high doses of low-molecular weight hydrocarbons is borne out by high dose studies in humans and animals for a number of different families of substances (Young, et al., 1977 [acrylonitrile]; DiVincenzo and Hamilton, 1979 [n-butanol]). Their applicability to the low exposure levels received in the environmental setting, however, is not clear.

For pesticides, a similar picture emerges. The levels of pesticide residues in foods and water have been well studied (Deinzer, *et al*., 1978; Kutz, *et al*., 1977). Pesticide residues have been measured in the fatty tissue, urine and feces of a large number of individuals during the first and second Health and Nutrition Examination Surveys (HANES) conducted by the National Center for Health Statistics, U.S. Public Health Service (Kutz, *et al*., 1977; 1978). The results of these studies are potentially very useful for identifying major sources of exposure and have even been able to identify time trends in the concentration of the residues of chlorinated organic pesticides and differences in pesticide residue levels between different racial and ethnic groups. However, they are of little use in elucidating exposure-dose relationships except on very general statistical levels, because simultaneous studies of individual exposures and dose have rarely been made at exposure levels typical of the general population.

### 4.2.2 Properties of Exposed Individuals and Populations Affecting Exposure-Dose Relationships

The study of populations and individuals that are highly susceptible to adverse health effects of pollutant exposure is reasonably well developed (Calabrese, 1978), and a review even of the most recent work in this field would be far beyond the scope of this chapter. There are, however, far fewer studies of the factors which predispose individuals and populations to absorb pollutants in an abnormal manner, as opposed to exhibiting enhanced responses to such exposure. The two fields, of course, overlap somewhat because one mechanism by which a similar pattern of exposure may produce reaction of greatly differing severity in two individuals is if they differ significantly in the amount of pollutant absorbed. The dose received by one individual will be higher than that received by the other.

In this section we briefly review recent research on factors which may contribute to differences in exposure-dose relationships among individuals and population groups. The major factors to be considered include primary demographic variables (age, sex and race), socioeconomic variables (occupation and income primarily), activity patterns and disease, and other abnormal physiological states which could affect pollutant absorption, including nutritional deficiencies where they are distinguishable from other socioeconomic variables. Other important factors which may affect the relationship between exposure (as measured by ambient pollutant concentration) and dose are activity patterns and other socioeconomic variables (e.g., residential patterns, income, etc.). These factors will affect exposure-dose relationships even if no differences in the efficiency of pollutant absorption exist.

### Primary Demographic Variables

A major focus of research in this area has been on the physiological differencies between infants, children, and adults. While it is well-known that most drugs can diffuse readily across the placenta from the maternal to the fetal circulation (Harbison, 1980), few data exist that would allow the identification of fetal characteristics that affect transport. The picture is further complicated by the greatly varying sensitivity of the fetus to teratogenic agents and other foreign substances at different stages of development.

Carbon monoxide can diffuse readily across the placenta, thus placing children of smoking mothers and women living in high-congested areas at higher risk. Heavy metal pollutants also can diffuse across the placenta, but there appears to be a modest protective effect against cadmium transfer (Buchet, *et al.*, 1978). Maternal and newborn blood levels of mercury and lead tend to be quite similar (indicating no barriers to diffusion exists) and an urban-rural gradient in maternal and fetal blood lead levels has been observed (Buchet, *et al.*, 1978).

Infants and children possess a number of physiological and behavioral characteristics that render them particularly liable to pollutant effects. Major behavior patterns that can be expected to contribute to greater uptake of pollutants by children are mouthing behavior or pica. They affect exposure to lead (absorbed from ingested paint chips) and to other pollutants commonly occurring in soil and household dust.

There are two physiological characteristics of children relative to adults that appear to have major effects on pollutant absorption. The first is a higher metabolic rate which manifests itself in the form of a more rapid breathing rate per unit weight in young children. It has been calculated, for example, that the ventilation rate (per body weight) for children 1-5 years of age is roughly twice that of adults (Knelsen, 1974). This would imply that children receive about twice the dose of any airborne particulate pollutant per unit body weight received by adults given similar ambient exposure levels. Behar (1979) has also pointed out how height gradients in the concentration of resuspended dust might well cause lead exposure experienced by children to be much higher than for adults. It is also to be expected that differences between pulmonary perfusion rates, amounts of adipose tissue and relative organ size in adults and children would affect the rate of absorption of gaseous pollutants (Fiserova-Bergerova, 1980).

The second physiological characteristic of children and infants affecting exposure - dose relationships is their tendency to absorb certain heavy metals (lead, cadmium, iron, cobalt) from the gastrointestinal tract much more efficiently than adults (Camner, 1979). This effect has been observed in humans for lead, where children absorb more than 50 percent ingested lead salts compared to 8-10 percent for adults. This effect is suspected to occur for the other metals on the basis of animal studies.

Recently an analysis has been performed of typical dietary patterns of different age groups in the United States with the intent of facilitating the analysis of pollutant body burdens (Rupp, 1979). Little is as yet known about the factors that affect dietary patterns for individuals as opposed to broadly defined socioeconomic and demographic groups.

Sex differences as determinants of pollutant dose have not been extensively studied. Differences in blood levels and body burden between males and females for a number of heavy metals have been observed (MITRE, 1979, 1979A; Camner, 1979), but the majority of these differences have been attributed to differences in exposure patterns. It is not known whether the enhanced uptake of iron during pregnancy has any effect on the absorption of other metals or toxics. Body burdens of organic chlorinated pesticides would be expected to be greater in women, who have a higher proportion of body fat than men. It is probable that this increased proportion of adipose tissue would also slightly enhance the rate of uptake of organic vapors through the lungs (Fiserova-Bergerova, 1981).

As in the case of sex differences, it appears as if the differences in body burdens of heavy metals (NAS, 1980) and pesticides (Kutz, _et al_., 1977) commonly observed between blacks and whites can be explained primarily as the basis of differences in exposure patterns and socioeconomic characteristics.

### Socioeconomic Characteristics

Socioeconomic characteristics act in many ways to influence the patterns of exposure to environmental pollutants, and indirectly, exposure-dose relationships. That is, despite the fact that there may be no differences in the physiological characteristics of one population compared to another, equal ambient concentrations of pollutant may have very different implications, in terms of absorbed dose, for the different groups. The socioeconomic aspects of environmental pollution exposure have been studied in great detail for environmental lead exposure. This serves in this section as a model for other pollutants, because one or more of the many socioeconomic variables which affect lead exposure-dose relationships have been found to apply to other pollutants as well.

A number of factors have been found to affect children's exposure-dose relationships for environmental lead. Among them are:

- Income and other socioeconomic variables that require certain groups to live in congested inner city areas where lead exposure from deteriorated lead paint is likely, where exposure to airborne particulate from vehicular traffic is likely to be high, and where lead pipes are more likely to be used to supply drinking water (EPA, 1977; NAS, 1980);
- Parental occupations which involve lead exposure may increase children's exposure to lead brought home on clothing or equipment (Rice, et al., 1978);
- Cultural and familial variables such as education level (parents of exposed children), access to medical information, number of children, birth order, characteristics of parent-child interaction, hygiene practices, and specific physical characteristics of the household (Stark, et al., 1978).

Similar socioeconomic determinants have been suggested to play a part in determining exposure to cadmium (MITRE, 1979a), and can be inferred for many industrial pollutants and pesticides (Kutz, et al., 1977). The clear implication of the findings so far are that minorities and the urban poor are generally at higher risk due to elevated pollutant intake. Unfortunately the models used to predict risk as a function of such variables are as yet still rather general and not sufficiently accurate to be used in identifying high-risk families and individuals from the general population (NAS, 1980).

## Activity Patterns and Physiological Variables

A known human activity that has the potential to alter the uptake and absorption of pollutants is smoking. Not only is smoking itself a major source of human exposure to cadmium (MITRE, 1979a) and an important determinant of indoor particulate levels (Repace and Lowrey, 1980), but the observation that long-time smokers may have reduced capacity to clear particles from the lung by ciliary transport implies that absorption of inhaled particles may be altered in these people. Such impaired clearance or altered absorption of particulate pollutants has not yet been demonstrated in practice and there is a wide degree of natural individual variation in the efficiency of mucociliary clearance, which is at least partially under genetic control (Camner, 1979).

Physical exertion and differences in respiratory rate can affect the intake of both particulate and gaseous pollutants through inhalation. Not only is total respiratory volume increased dramatically during strenuous exercise, but the efficiency of particle deposition changes with rapid breathing. High flow rates in the nasopharynx and trachebronchial regions can increase the efficiency of deposition of large (≥ 1 micron mass median aerodynamic diameter (MMAD)) particles in these regions (Camner, 1979), while mouth breathing during heavy exercise decreases particle deposition to an unknown extent.

Rapid respiration and increased pulmonary perfusion during exercise should increase the rate of uptake for many gaseous pollutants (Klaasen, 1980, Fiserova-Bergerova, 1980), although it is not clear that the total absorbed dose would be increased under these circumstances. One recent study of methylene chloride exposure during exercise did find an increase in total uptake, partially driven by the increased rate of metabolism of the substance that was found to occur (DiVincenzo and Kaplan, 1981). The relevance of this observation for environmental exposure is not clear, although there is sufficient grounds to suspect that activity levels may, for the reasons discussed above, be an important determinant of exposure-dose relationships for some populations.

The last major topic to be discussed in this section is the complex relationship between nutritional status and the uptake of certain pollutants. The primary effect in this case appears to be on the absorption of heavy metals, which is not only affected by general nutritional status but by specific pollutant-pollutant and pollutant-nutrient interactions.

Lead absorption is known to be enhanced in individuals receiving low levels of essential nutrients such as calcium, iron and zinc (Mahaffey and Vanderveen, 1979). Absorption of lead is also elevated by the presence of casein (milk protein) and chelating agents in the diet. The former observation is thought by some to be partially responsible for the elevated absorption of lead by infants. The efficiency of human absorption of dietary cadmium is enhanced by diets low in calcium or protein, and there appears to be a complex relationship between cadmium uptake and dietary zinc concentration, with zinc showing some protective effect against the adverse effects of cadmium exposure (MITRE, 1979a). For the toxic elements mercury and arsenic, less is known about nutrient-toxicant interrelations, although the presence of pentavalent arsenic salts may affect the rate of uptake of toxic selenium salts (Venogupal and Luckey, 1976). For many of the metals which are essential at low concentration (e.g., zinc, copper, iron), there are homeostatic mechanisms which can control uptake within certain limits.

There are reports that uptake of certain pesticides are also affected by nutritional status. For example, the gastrointestinal absorption of dieldrin (Klaasen, 1980) and heptachlor (Mahaffey and Vanderveen, 1979) were increased in starved laboratory animals. Such results have not been directly observed in humans.

## 4.3 PHYSIOLOGICAL INDICATORS OF DOSE

In this section we discuss recent research concerning the development and use of physiological indicators of pollutant exposure and dose. Such indicators usually take the form of measurements of pollutants or metabolite concentrations in body fluids, tissues or exhaled air. The rationale behind the development of such indicators is that they can be used to provide information about the intensity of recent exposures and doses of pollutant and help to identify important sources and routes of exposure.

As is often the case, lead is the most well-studied example and the paradigm for other pollutants. Studies on the relationship between air, water and food-borne exposures to lead and blood lead levels began in the 1930's (Kehoe, 1961). By the middle 1970's numerous epidemiologic studies of the relationships between blood lead and air lead exposure had been performed, both in the occupational and environmental settings (NAS, 1972; EPA, 1977). Several models had been developed which describe the relationship between blood lead levels and total absorbed dose of lead (MITRE, 1979) in the form of simple linear relationships. Such models seem to be reasonably useful in describing the relationship between average blood lead levels and recent exposure for large populations with "typical" physiological characteristic where exposures have not changed over time. They are not so useful, however in constructing detailed exposure histories for individuals, or where recent exposures have changed dramatically. This is because blood lead levels respond somewhat slowly (over a period of several months) to changes in exposure. Blood lead levels do not correlate well with total body burden, which after long-term exposures becomes deposited primarily in the bones. Blood lead levels also do not correlate well with the severity of chronic toxic effects (NAS, 1980). At a first approximation, however, there may be defineable blood lead levels below which certain adverse effects do not occur. Also, very high blood lead levels are good indicators of the potential for acute toxic effects (EPA 1977).

Other indicators of lead exposure and absorption have been investigated. Urinary lead excretion correlates less well with recent exposures than blood lead level, but measurements of the activity of certain enzymes and metabolites in the blood seem to correlate well with recent exposures. The enzyme, α-aminolevulinic acid dehydratase (ALAD), an important component in the heme synthetic pathway; α-aminolevulinic acid (ALA), a related metabolite which builds up in the blood because of the reduction in ALAD; and erythrocyte protoporphyrin (EP) have been found to correlate well with blood lead levels above about 15-20 μg/100cc and thus provide reasonably sensitive indices of lead absorption. EP is considered to be the most useful indicator of lead exposure because it is closer than ALAD or ALA to the end product of the synthetic pathway, namely hemoglobin (EPA 1977). Because lead inhibits the synthesis of hemoglobin, high enough exposure may produce anemia, which in itself may be a physiologic indicator of lead exposure.

As mentioned earlier, body burden of lead is not closely correlated with either recent lead exposure or the magnitude of chronic toxic effects. Similary, attempts to correlate lead levels in brain, neural tissue and kidney, major target organs for lead toxicity, do not generally provide useful (or practical) indicators of either lead dose or adverse health effects.

In the case of cadmium, the picture is somewhat less complex. Because of the relatively rapid clearance of cadmium from the body, measurements of cadmium levels in blood, urine, or hair do not provide accurate estimates of recent exposures in situations where exposures have changed over time in any significant fashion (MITRE, 1979a). Where exposure has been constant, blood and urine levels should, in theory provide reasonably good indices of exposure levels (Kjellstrom and Nordberg, 1978). The concentration of cadmium in the kidney (a major target for toxic effects) provides a reasonably good indication of the likelihood and severity of adverse effects (MITRE, 1979a; Camner, 1979).

The picture for mercury is complicated by the fact that inorganic and organic mercury compounds (most usually methylmercury and its derivatives) behave quite differently in the body. Inorganic mercury salts are cleared from the body with a half-life of about 40 days. They are carried about equally in plasma and red cells and deposited almost exclusively in the kidney. Methylmercury is cleared from the body with a half-life of about 70 days, and is carried mostly in red blood cells and deposited in the brain and kidneys. Blood mercury levels seem to be a reliable indicator of exposure as long as exposure levels have been relatively constant for several months, but less reliable if recent high exposures have occurred or if exposure is intermittent (Kershaw, 1980). Unlike lead and cadmium, it appears that the total body burden of mercury may be a useful predictor of at least some of the more quantifiable neurological symptoms (Bakir, et al., 1973).

We have already briefly discussed the relationship between exposure to organic vapors and the uptake of these substances. We noted how, for most volatile organics, the attainment of equilibrium between the concentrations of the substance in the inhaled and exhaled air and tissue occurred relatively rapidly under laboratory conditions, and how concentrations of vapor in exhaled air decreased rapidly after the cessation of exposure. (Fiserova - Bergerova, 1980, 1981; DiVincenzo and Kaplan, 1981). Thus, for compounds that are not metabolized to distinctive products, the use of the measurements of volatile organic compounds in expired air or blood would usually be a poor index of recent exposures. It would almost always be more practical to simply measure ambient air levels. If, however, the major route of exposure to such compounds were by some route other than inhalation, e.g., by ingestion, such measurements might gain new relevance as indices of total exposure. One recent study of exposure to volatile organic hydrocarbons in members of the general population has investigated the possibility of using measurement of levels of volatile chlorinated organics in blood and exhaled air as measures of total exposure (Wallace and Pellizzari, 1982). Preliminary results have been encouraging for breath but not for blood. It appears as if analysis of exhaled air can be used not only as an index of exposure to pollutants primarily absorbed from air (1,1,1-trichloroethylene and perchloroethylene) but also may prove useful in the case of exposure to pollutants taken up primarily from drinking water (chloroform). A model relating breath concentrations to previous breath and air concentrations was created and parameters for uptake and effective biological half-life were estimated for the two atmospheric chemicals mentioned above.

Two commonly occurring compounds, methylene chloride and trichloroethylene, are metabolized to produce relatively long-lived and distinctive metabolites that can be used to characterize recent exposures over a time frame of several hours to a few days. Relatively high concentrations (50-200 ppm) of methylene chloride, for example, are rapidly metabolized to carbon monoxide which can be measured as an increase in the concentration of carboxyhemoglobin (hemoglobin with carbon monoxide bound to it) for as long as 16-25 hours after exposure (DiVincenzo and Kaplan, 1981). Such high concentrations are rarely encountered in the environment, however. Trichloroethylene is metabolized to trichloroacetic acid and trichloroethanol upon inhalation (Fernandez, et al., 1977). The former has a half-life of several days in the blood stream; the latter, about a day. Thus both of these compounds might be useful as indicators of dose over a fairly long period of time. Few other volatile organic compounds commonly found in the environment possess similarly distinctive metabolites.

A thorough review of the literature on the persistence of pesticide residues in the human body is beyond the scope of this chapter. Measurement of the residues of halogenated organic and phosphate pesticides in large populations have been used as indicators of the prevalence, as well as the magnitude, of pesticide exposure. Analysis of these data have allowed investigators to study the time trends in the relative degree of exposure of the general population and have demonstrated that higher average levels of pesticide residues occur in black Americans then in whites (Kutz, et al., 1977).

## 4.4 MODELS FOR THE EVALUATION OF EXPOSURE-DOSE RELATIONSHIPS AND THE INTEGRATION OF MULTISOURCE EXPOSURES

This section discusses two types of mathematical models used in analysis of the relationships between exposure and dose for environmental pollutants. The first type of model considered is the classical pharmacokinetic model describing the uptake, transport, metabolism and excretion of pollutants from the body. A large number of such models exist for many individual pollutants. These models are discussed only to the extent that such discussion is relevant to the understanding of exposure-dose relationships, and to give the reader some idea of the scope of the literature in the area.

The second type of model discussed are those which are designed to integrate exposures to more than one pollutant or by more than one route of exposure. These models, of which there are relatively few, are much more relevant to the issue of total exposure and dose assessment.

Classical pharmacokinetic models have been developed for most of the major toxic metals. There are two well developed models for the metabolism of inorganic lead, each of which has more than one variant (Marcus, 1979). They postulate either three or five pharmacokinetically distinct compartments and both models can be used to predict the total body burden of lead fairly well compared to experimental data. All of these models use relatively crude assumptions about the relationships between exposure and dose, even though this relationship has been shown to be very important (Mendez and Hattis, 1978). Models also exist for cadmium (Kjellstrom and Nordberg, 1978), mercury (Nordberg and Strangert, 1978; Bakir, 1973), and selenium (Medinsky, et al., 1981). The International Committee on Radiation Protection has developed a family of general models which describe the persistence and elimination of radionuclides from the body (ICRP, 1966), which are continually revised for specific substances as additional data are developed. In all of these models, as in the case of lead, the assumptions about exposure-dose relationship are exogenous to the pharmacokinetic models themselves. Thus, as much as anything else, these substance-specific models are useful tools for analyzing alternative assumptions about exposure-dose parameters.

Models for the uptake, metabolism and excretion of volatile organic chemicals are generally more firmly grounded in experimental data relating to the uptake of these substance by human subjects. The family of models developed to analyze the behavior of these substances in the body (Fiserova-Bergerova, 1980, 1981) has been applied to the analysis of eight volatile organic chemicals including benzene, trichloroethylene, methylene chloride and halothane. These results were then verified against experimental data by the original investigators and confirmed by others (DiVincenzo and Kaplan, 1981; Peterson, 1978). Unlike the models describing the pharmacokinetic behavior of metals, these models incorporate observational data about the uptake of the substances as integral components. Many of the general predictions made by these models about equilibrium levels of volatile organic chemicals in blood, inhaled air and exhaled air seem to be confirmed in the environmental setting by the preliminary results of several studies (Wallace and Pellizzari, 1982;

Wallace, 1983). None of these models, however, include features which allow for the analysis of the effect of significant intake of chemicals by routes other than inhalation (e.g., by ingestion of polluted drinking water). For some substances, these routes provide a major source of exposure.

The final type of pharmacokinetic models that deserve mention are those which relate the concentration of carboxyhemoglobin in the blood to recent carbon monoxide exposures. The relationships between the intensity and duration of carbon monoxide exposures and levels of carboxyhemoglobin have been well studied and the kinetics of the binding of carbon monoxide to hemoglobin and its displacement have been elucidated (Coburn, 1970). Recently, a simple computer model has been developed which simulates the effects of carbon monoxide exposures on carboxyhemoglobin levels (Ott and Mage, 1978). This model has been used to evaluate the implications of the National Ambient Air Quality Standard for carbon monoxide and has provided useful information about the averaging periods which might be appropriate for monitoring ambient carbon monoxide concentrations. Another such model (Joumard, et al., 1981) has been used to study the effects of age, sex and physiological differences between individuals on the levels of carboxyhemoglobin in the blood. It has also been used, with a simple behavioral model, to analyze weekly patterns in carboxyhemoglobin levels.

All of these models, while they may be very useful in analyzing the implications of specified exposure and dose levels, may not be particularly useful in integrating exposures from different sources or in characterizing "total exposures" and doses arising from complex exposure scenarios. The models usually take, as given, assumptions about the relationships between exposure patterns and doses.

The next, smaller family of models we discuss take a different approach, and are specifically designed to be used directly or indirectly to model the relationships between complex exposures and doses. In some cases these models consist merely of a set of weighting factors which define how contributions from exposures in different media are to be counted as contributing to total dose or body burden.

The first model we discuss is intended to be used to develop exposure limits for pollutants in the ambient environment that will limit the concentration of pollutant in the body or in a target organic to less than a selected value, when exposures from all sources are taken into account (Walsh, et al., 1978). The approach taken is to develop equations which define the processes by which exposure through each important route occur using steady-state assumptions about the rate of pollutant transfer between the various media, and between the environment and the body. For example, the limiting concentration in air (Ca) for a pollutant that is emitted into the air would be:

$$C_a = Q_1 \left[ \left( V_a F_r + F_{af} M_f F_d + F_{aw} \left( V_w F_d + E_w K_w F_s \right) + K_a F_s \right) R \right]^{-1}$$

where $Q_1$ is the limiting concentration in the body or organ of interest, $V_a$ is the volume of air inhaled, $F_r$ is the "transfer coefficient" for absorption of the pollutant through inhalation, $F_{af}$ is the transfer factor for air-to-food transfer, $M_f$ is daily food intake, and $F_d$ is the transfer factor for the absorption of ingested pollutants. The remaining terms describe the transfer of pollutant for air to water ($F_{aw}$), the uptake of pollutant from drinking water ($V_wF_d$) and through dermal contact ($E_wK_wF_s$) and exposure through contaminated soil ($K_aF_s$). R is the steady-state retained fraction of pollutant in the body or organ. The transfer coefficients are all defined so that they fulfill the condition $C_i = F_{ij}C_j$ that is, they define the steady-state ratios of concentration between different media.

Where multiple sources and routes contribute to total exposure, the equation for limiting concentration in any one medium becomes much more complex, since transfers between the various media could occur in both directions. In many cases, however, intermedia transfers can be neglected, greatly simplifying the model.

This model has been used to define a limiting concentration for cadmium in air near a smelter (Rupp, et al., 1978). While the results appear to be quite reasonable and much useful information was generated, this example points up the major weakness with the model. This weakness is the need to express complex dynamic processes (air dispersion of cadmium particulate, uptake and pharmacokinetic-distribution) in terms of simple steady-state concentration ratios. Even with this problem, the general approach to mathematically defining complex exposures seems quite promising even though it was not intended for such use. What is needed is a better understanding of when the steady state assumptions are valid and how seriously their violation affects the accuracy of the model. Some of these issues have been addressed (Drake, 1980) and attempts have been made to model the rate at which intermedia pollutant transfer actually occurs.

A similar approach to analyzing contributions to exposure and dose from multiple sources and pathways has recently been developed (Rosenblatt, et al., 1980). The approach differs principally in that, instead of using intermedia transfer coefficients calculated from specific studies of pollutant dispersion, it seems to advocate the use of estimated partition coefficients as an approximation for the ratio of concentrations of pollutants in the various media. A "conservative" value of 1.0 is used when the observed value is not available. Use of this model could impose yet another level of uncertainty on the exposure assessment since "steady state" concentration ratios may differ from equilibrium partitioning between media. However, the authors do point out the desirability of the use of transfer coefficients where they are available, so that, where good data are available, the results from their model and the previously described model should agree closely.

Neither of these two models, which seem to be the most comprehensive and ambitious available for analyzing the relative contribution of various sources and routes of exposure to "total exposure," are capable of analyzing dynamic, non-steady-state situations. Neither includes features which could take into account the attenuation or degradation of pollutants in the environment, although such features could probably be added fairly easily. This makes either of the models a useful tool for analyzing total exposure.

In addition to these complex, comprehensive mathematical models for the integration of exposure, there are a number of much simpler "models," some of which are merely rules for weighting exposures by various media to assess total exposures. One such model has been used to calculate relative contributions to lead intake for children, pregnant women, and adult males (MITRE, 1979) and to perform similar calculations for cadmium exposure (MITRE, 1979a). These studies employ the standard assumptions about uptake efficiency of airborne particulate and absorption lead in the lungs and gastrointestinal tract. While the general approach is useful in clarifying relative contributions to dose levels under various scenarios, the information generated is only as useful as the quality of the assumptions employed. One interesting result is the very significant contribution of soil and dust to the total lead exposure of children without pica, which may suggest useful and practical methods for dealing with the problem of lead toxicity in children.

## 4.5 RESEARCH NEEDS

In our analysis of recent literature of exposure-dose relationships, a number of important information gaps and important areas for future research came to our attention.

One of our major findings was that while many of the basic physiological factors that affect the uptake of pollutants by humans have been extensively studied and general models seem to exist, very little is known about the sources and magnitude of individual variations in these factors that might occur within defined populations. Some of these factors are poorly understood, even at a statistical level, in many instances. Predictions about specifically defined populations must reference one or another of the few standard models for pollutant uptake, perhaps with some simple correction factors when something is known about the particular group under study. Clearly, more detailed studies of the sources of variations in pollutant uptake between individuals and by individuals over time are required. The study of groups where abnormal responses to pollutants arise from altered patterns of absorption, for example, is still in its infancy. The data base about known physiological responses to pollutants is still predominantly made up of data about adults. While the unique physiological properties of children which affect pollutant uptake have been studied in a few cases, relatively few data are not simply derived by analogy to studies in adults.

The use of physiological indicators of dose is a rapidly expanding area of research that could be greatly facilitated by improvements in and applications of pharmacokinetic models. Such models for environmental pollutants as they exist now in the literature, tend to emphasize metabolism and transport within the body and regard considerations of uptake efficiency as being of secondary importance. Much more work is still required in order to make many of these models useful in assessing the health impacts of different exposure patterns relevant to public policy choices.

The initial attempts at developing comprehensive models for total exposure and dose assessment have been aimed primarily at setting acceptable exposure limits. The theoretical basis for more generalizable models needs to be investigated. The entire area of exposure-dose analysis, requiring, as it sometimes does, rather crude assumptions and extrapolation from limited data, does not appear to be an attractive area of effort for many investigators, despite the fact that the analysis of important public policy issues could be facilitated by such research.

# LIST OF REFERENCES

Ashford, N.A., D. Hattis, F.R. Heaton, J.I. Katz, W.C. Priest, and E.M. Zolt. Evaluating Chemical Regulations: Trade-off Analysis and Impact Assessment for Environmental Decision-Making. Center for Policy Alternatives, Massachusetts Institute of Technology, Cambridge, Mass, 1980.

Bakir, F., et al., Methylmercury Poisoning in Iraq. Science, 181:230-41, 1973.

Behar, J. V., E. A. Schuck, R. E. Stanley, and G. B. Morgan. Integrated Exposure Assessment Monitoring. Environmental Science and Technology, 13:34-39, January 1979.

Buchet, J. P., H. Roels, G. Hubermont, and R. Lauwerys. Placental Transfer of Lead, Mercury, Cadmium, and Carbon Monoxide in Women. Part II. Influence of Some Epidemiological Factors on the Frequency Distributions of the Biological Indices in Maternal and Umbilical Cord Blood. Environmental Research, 15:494-503, 1978.

Calabrese, E.J. Pollutants and High-Risk Groups, J. Wiley, New York, 1978.

Camner, P., et al. Routes of Exposure, Dose and Metabolism of Metals. Handbook on the Toxicology of Metals, Elsevier, New York, 1979.

Carcinogen Assessment Group. Method of Determining Unit Risk Estimates for Air Pollutants. U.S. Environmental Protection Agency, July 1980.

Coburn, R.F., editor. Biological Effects of Carbon Monoxide. Annals of the New York Academy of Sciences, 1974, 1970.

Colome, S.D., S. McCarthy, and J.D. Spengler. Residential Indoor and Ambient Outdoor Comparison of Gaseous and Particulate Air Pollutants in Two Cities. 74th Annual Meeting of the Air Pollution Control Association, Philadelphia, PA, June 21-26, 1981.

Colwill, D. M. and A. J. Hickman. Exposure of Drivers to Carbon Monoxide. Journal of the Air Pollution Control Association, 30(12):1316-1319, December 1980.

Cortese, A.D. Ability of Fixed Air Monitoring Stations to Represent Personal Personal Carbon Monoxide Exposures. Ph.D. Dissertation, Harvard University, School of Public Health, 1976.

Deinzer, M., F. Schaumburg, and E. Klein. Environmental Health Sciences Center Task Force Review on Halogenated Organics in Drinking Water. Environmental Health Perspectives, 24:209-239, June 1978.

Dennis, R. L. An Air Pollution Dispersion Model for Long-Range Policy Analysis. Ecological Modeling, 2:121-141, March 1980.

DiVincenzo, G.D. and M.L. Hamilton. Fate of n-Butanol in Rats after Oral Administration and Its Uptake by Dogs after Inhalation or Skin Application. Toxicology and Applied Pharmacology, 48:317-325, 1979

DiVincenzo, G.D., and C.J. Kaplan. Effect of Exercise and Smoking on the Uptake, Metabolism and Excretion of Methylene Chloride Vapor. Toxicology and Applied Pharmacology, 49:141-148, 1981.

Dockery, D. W. and J. D. Spengler. Personal Exposure to Respirable Particulates and Sulfates. Air Pollution Control Association Journal, 31:153-159, 1981.

Drake, R. L. (Battelle Pacific Northwest Laboratory). Interfacing Air Pathway Model with Other Media Models for Impact Assessment. Presented at the 20th Hanford Life Sciences Symposium, Richland, Washington, October 19-23, 1980. U. S. Department of Energy Contract No. DE-AC06-76RLO-1830, October 1980.

Eifler, C. W., S. S. Stinnett, and P. A. Buffler. Simple Models for Community Exposure from Point Sources of Pollution. Environmental Research, 25:139- 146, 1981.

Fernandez, J., et al. Trichloroethylene Exposure -- Simulation of Uptake, Excretion and Metabolism, Using a Mathematical Model. British Journal of Industrial Medicine, 34:43-55, 1977.

Fiserova-Bergerova, V. Predicting "Individual Differences" in Uptake and Excretion of Gases and Lipid Soluble Vapors: Simulation Study. British Journal of Industrial Medicine, 31: 45-52, 1974.

Fiserova-Bergerova, V. Predicting "Individual Differences" in Uptake and Excretion of Gases and Lipid Soluble Vapors: Simulation Study. British Journal of Industrial Medicine, 37:42-49, 1980.

Fiserova-Bergerova, V. Modeling of the Uptake and Clearance of Inhaled Vapors and Gases. Industrial and Environmental Xenobiotics, I. Gut, et al., editors, Springer-Verlag, New York, 1981.

Gasper, J. R. and P. A. Dauzvardis. (Argonne National Laboratory, Argonne, Illinois) Body Burdens--An Integrated Approach to Health Risk Assessment. Prepared for 1979 National Conference on Hazardous Material Risk Assessment, Disposal and Management, Miami Beach, Florida, April 25-27, 1979. 18 pp.

Good, B. W., G. Vilcins, *et al*. Effect of Cigarette Smoking on Residential $NO_2$ Levels. *Environment International*, 8:167-183, 1982.

Harbison, R.D. Teratogens. *Toxicology: The Basic Science of Poisons*, J. Doull, *et al*., editors, Second edition, MacMillan, Toronto, 1980.

Hedden, K.F. Water Treatment Model for Pollutant Exposure Assessment. *Journal of Environmental Science and Health-Environmental Science and Engineering*, A15:285-305, 1980.

Hickey, J. L. S. and P. C. Reist. Adjusting Occupational Exposure Limits for Moonlighting, Overtime, and Environmental Exposures. *American Industrial Hygiene Association Journal*, 40:727-733, 1979.

International Commission on a Radiation Protection, Task Group on Lung Dynamics. Deposition and Retention Models for Internal Dosimetry of the Human Respiratory Tract. *Health Physics*, 12:173, 1966.

Jabara, J.W., *et al*. Carbon Monoxide: Dosimetry in Occupational Exposures in Denver, Colorado. *Archives of Environmental Health*, 35:198-206, 1980.

Joumard, R., *et al*. Mathematical Models of the Uptake of Carbon Monoxide on Hemoglobin at Low Carbon Monoxide Levels. *Environmental Health Perspectives*, 41:277-290, 1981.

Kehoe, R.A. Toxicological Appraisal of Lead in Relation to the Tolerable Concentration in the Ambient Air. *Journal of the Air Pollution Control Association*, 19:960-701, 1969.

Kehoe, R.A. The Harben Lectures. *Journal of the Royal Institute of Public Health*, 24:81-97, 1961.

Kershaw, T.G. The Relationship Between Blood Levels and Doses of Methyl Mercury in Man. *Archives of Environmental Health*, 35:28-36, 1980.

Kjellstrom, T. and Nordberg, G. A Kinetic Model of Cadmium Metabolism in the Human Being. *Environmental Research*, 16:248-269, 1978.

Klaasen, C.D. Absorption, Distribution and Excretion of Toxicants. *Toxicology: The Basic Science of Poisons*, J. Doull, *et al*., editors, Second edition, MacMillan, Toronto, 1980.

Knelson, J.H. Problems of Estimating Respiratory Lead Dose in Children. *Environmental Health Perspectives*, 35:53-58, 1974.

Kutz, F. W., S. C. Strassman, A. R. Yobbs, and J. F. Viar, Jr. Pesticides in People. Pesticides Monitoring Journal, II:61-63, September 1977.

Kutz, F. W.,, S. C. Strassman and J. F. Sperling. Survey of Selected Inorganic Pesticides in the General Population of the United States. Annals of the New York Academy of Sciences, 320:60-68, May 31, 1978.

Mahaffey, K. R. and J. E. Vanderveen. Nutrient-Toxicant Interactions: Susceptible Populations. Environmental Health Perspectives, 29:81-87, April 1979.

Medinsky, M., et al. A Simulation Model Describing the Metabolism of Inhaled Selenium Compounds. Toxicology and Applied Pharmacology, 59:54-63, 1981.

Mendez, W. M., Jr. and D. Hattis. Analysis of Available Evidence on Blood Lead-Air Lead Relationships Relevant to the Selection of a Permissable Exposure Limit for Lead in Air. Center for Policy Alternatives, Massachusetts Institute of Technology, Cambridge, Massachusetts, CPA-78-13, June 1978.

Menzel, D.B. and R.O. McClellan. Toxic Responses of the Respiratory System. in Toxicology: The Basic Science of Poisons, J. Doull, et al., editors, Second edition, MacMillan, Toronto, 1980.

Miller, C. Exposure Assessment Modeling: A State-of-the-Art Review. EPA-600/3-78-065, U. S. Environmental Protection Agency, Environmental Research Laboratory, Office of Research and Development, Athens, Georgia, July 1978.

The MITRE Corporation. The Environmental Lead Problem: An Assessment of Lead in Drinking Water from a Multi-Media Standpoint. EPA-570/9-79-003, U.S. Environmental Protection Agency, Criteria and Standards Division, 1979.

The MITRE Corporation. An Assessment of Cadmium in Drinking Water from a Multi-Media Perspective, 1979a.

Morrow, P.E. Pulmonary Retention of Lead in Man: An Experimental Study. Environmental Research, 21:373-384, 1980.

Moschandreas, D. J. and Stark, J. W. (GEOMET, Inc.) The Residential Environment and Energy Conservation: Predicting Indoor Air Quality. Presented at the 71st Annual Meeting of the Air Pollution Control Association, Houston, Texas, June 25-30, 1978. 14 pp.

Moschandreas, D. J. Indoor Air Pollution in the Residential Environment--Volume II: Field Monitoring Protocol, Indoor Episodic Pollutant Release Experiments and Numerical Analyses. EPA-600/7-78-229b, U.S. Environmental Protection Agency, Office of Research and Development, Environmental Monitoring and Support Laboratory, Research Triangle Park, North Carolina, December 1978. 231 pp.

Nagda, N. L. (GEOMET, Inc.) Environmental Carcinogens and Human Cancer--Estimation of Exposure to Carcinogens in the Ambient Air. EPA-600/1-79-002. U.S. Environmental Protection Agency, Office of Research and Development, Health Effects Research Laboratory, Research Triangle Park, North Carolina, 1979. 155 pp.

National Academy of Sciences, National Research Council, Committee on Biological Effects of Atmospheric Pollutants. Lead: Airborne Lead in Perspective, Washington, D.C., 1972.

National Academy of Sciences, Committee on Lead in the Human Environment. Lead in the Human Environment, Washington, D.C., 1980.

National Academy of Sciences. Drinking Water and Health, Washington, D.C. 1977.

Nitta, H. and K. Maeda. Personal Exposure Monitoring to Nitrogen Dioxide. Environment International, 8:243-248, 1982.

Nordberg, G. and P.Strangert. Fundamental Aspects of Dose-Response-Relationships. Environmental Health Perspectives, 22:97-102, 1978.

Ott, W. An Urban Survey Technique for Measuring Spacial Variation of Carbon Monoxide Concentrations in Esters. Ph.D. Dissertation, Stanford University, Department of Civil Engineering, 1971.

Ott, W. R. Exposure Estimates Based on Computer Generated Activity Patterns. Prepared for presentation at the 74th Annual Meeting of the Air Pollution Control Association, Philadelphia, Pennsylvania, June 21-26, 1981.

Ott, W. R. Models of Human Exposure to Air Pollution. Technical Report No. 32, July 1980. Prepared under the auspices of Siam Institute for Mathematics and Society.

Ott, W. and P. Flachsbart. Measurement of Carbon Monoxide Concentrations in Indoor and Outdoor Locations Using Personal Exposure Monitors. Environment International, 8:295-304, 1982.

Ott, W.R. and Mage, D.T. Interpreting Urban Carbon Monoxide Concentrations by Means of a Computerized Blood COhb Model. Journal of the Air Pollution Control Association, 28:911-916, 1978.

Pellizzari, E.D., et al. Total Exposure Assessment Methodology (TEAM) Study: Phase II, Part II: Protocols for Environmental and Human Sampling and Analysis. Office of Monitoring Systems and Quality Assurance, U.S. Environmental Protection Agency. 1981.

Peterson, J.E. Modeling the Uptake, Metabolism and Excretion of Dichloromethane by Man. American Industrial Hygiene Association Journal, 39:41-47, 1978.

Repace, J. and A. H. Lowrey. Indoor Air Pollution, Tobacco Smoke, and Public Health. Science, 208:464-472, 1980.

Rice, C., et al. Lead Contamination in the Homes of Employees of Secondary Lead Smelters. Environmental Research, 15:375-380, 1978.

Rosenblatt, David H. and David R. Cogley. An Environmental Fate Model Leading to Preliminary Pollutant Limit Values for Human Health Effects. Environmental Risk Analysis for Chemicals, Richard Conway, ed., Van Nostrand Reinhold Company, New York, 1980.

Rupp, E. M. Age Dependent Values of Dietary Intake for Assessing Human Exposures to Environmental Pollutants. Health Physics, 39:151-163, 1979.

Rupp, E. M., et al. Composite Hazard Index for Assessing Limiting Exposures to Environmental Pollutants: Application through a Case Study. Environmental Science and Technology, 12:802-807, 1978.

Rydell, C. P., D. Collins, A. Dedrick, H. Kaminski, and G. Schwarz. Air Pollution and Urban Population Distribution. Hunter College Research Center, CUNY, under U.S.Public Health Service Grant No. AP 00555, July 1968.

Stark, A.D., et al. Family Operation Cofactors in the Epidemiology of Childhood Lead Poisoning. Archives of Environmental Health, 33:222-226, 1978.

Todd, W.J. et al. Satellite-Aided Evaluation of Pollution Exposure to Air Pollution. Environmented Science and Technology, 13: 971-174, 1979.

U.S. Environmental Protection Agency. Air Quality Criteria for Lead. EPA-600/8-77-017, U.S. Environmental Protection Agency, Office of Research and Development, Washington, D. C., December 1977.

U.S. Environmental Protection Agency. Analysis of Population Exposure in New York - New Jersey - Connecticut Tri-State Region. EPA-450/3-76-027. U.S. Environmental Protection Agency, Office of Air and Waste Management, Office of Air Quality Planning and Standards, Research Triangle Park, North Carolina, March 1976.

U.S. Environmental Protection Agency. National Interim Primary Drinking Water Regulations; Control of Trihalomethanes in Drinking Water. 44 Federal Register 68624-68706, November 29, 1979. (40 CFR Part 141)

U.S. Environmental Protection Agency. The Carcinogenic Assessment Group's Final Risk Assessment on Arsenic. EPA 600/6-81-002, U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards, Washington, D. C., 1981a.

U.S. Environmental Protection Agency. Draft Research Plan for Monitoring Human Exposure to Environmental Pollutants in All Media. Prepared by the Office of Monitoring Systems and Quality Assurance. 1981b.

U.S. Environmental Protection Agency. Graphical Exposure Assessment Model (GEMS) User's Guide, Office of Pesticides and Toxic Substances, Exposure Evaluation Division, December 19, 1982.

U.S. Environmental Protection Agency. Handbook for Performing Exposure Assessments. Compiled by Exposure Assessment Group, Office of Health and Environmental Assessment. 1981c.

U.S. Environmental Protection Agency and U.S. Department of Energy. Inventory of Current Indoor Air Quality-Related Research. Prepared for the U.S. Environmental Protection Agency, Office of Research and Development, Industrial Environmental Research Laboratory, Cincinnati, Ohio, 1981d.

U.S. Environmental Protection Agency. A Research Plan for Population Exposure Monitoring Methodology: Vehicular Air Pollutants. Prepared by the Office of Monitoring Systems and Quality Assurance and the Office of Mobile Source Air Pollution Control. 1981e.

U.S. Environmental Protection Agency, et al. Summary of the Workshop on Exposure to Environmental Agents, Their Metabolism, and Mechanisms of Toxicity--Research Needs. Prepared by the Project Group on Exposure and Metabolic Mechanisms, Task Force on Environmental Cancer and Heart and Lung Disease. 1981f.

U.S. Environmental Protection Agency. Standards of Performance for New Stationary Sources: Volatile Organic Compounds (VOC) Fugitive Emission Sources in the Synthetic Organic Chemicals Manufacturing Industry. 46 Federal Register 1136-1165, 1981g.

U.S. Occupational Safety and Health Administration. Occupational Exposure to Lead, Attachment to the Final Standard. 43 Federal Register 54353-54616, November 21, 1978.

Venogupal, B., and T.D. Luckey, Metal Toxicity in Mammals, Volume 2. Plenum Press, New York, 1976.

Verberk, M.M. and T.M.L. Scheffers. Tetrachloroethylene in Exhaled Air of Residents Near Dry Cleaning Shops. Environmental Research, 21:432-437, 1980.

Wallace, L. Individual Human Exposures to Volatile Organic Compounds Encountered During Normal Daily Activities. Submitted to Environmental Research, 1983.

Wallace, L. Use of Personal Monitors to Measure Commuter Exposures to Carbon Monoxide in Vehicle Passenger Compartments. Paper 79-59.2 presented at 72nd Annual Meeting of APCA, 1979.

Wallace L., and W. Ott. Personal Monitors: A State-of-the-Art Survey. Journal of the Air Pollution Control Association, 32:601-610, 1982.

Wallace, L. and Pellizzari, E.D. Direct Measurements of Volatile Organic Compounds in Breathing Zone Air, Drinking Water, Breath and Blood. Environment International, 8, 1982.

Walsh, P. J., et al. Composite Hazard Index for Assessing Limiting Exposures to Environmental Pollutants: Formulation and Derivation. Environmental Science and Technology, 12:799-802, 1978.

Yanagisawa, Y. and H. Nishimura. A Badge-Type Personal Sampler for Measurement of Personal Exposure to $NO_2$ and NO in Ambient Air. Environment International, 8:235-242, 1982.

Yocum, J.E. "Indoor-Outdoor Air Quality Relationships. A Critical Review. Journal of the Air Pollution Control Association, 32:500-520, 1982.

Young, J.E., et al. The Pharmacokinetic and Metabolic Profile of $^{14}C$-Acrylonitrile Given to Rats by Three Routes. Prepared by the Toxicology Research Laboratory, Health and Environmental Research, Dow Chemical Company, Midland, MI., U.S.A. for the U.S. Environmental Protection Agency, 1977.

Zweidinger, R., et al. Monitoring Individual Exposure--Measurements of Volatile Organic Compounds in Breathing-Zone Air, Drinking Water, and Exhaled Breath. U.S. Environmental Protection Agency, Office of Monitoring Systems and Quality Assurance, Office of Research and Development, 1981.

B-1.a

# TOTAL EXPOSURE ANNOTATED BIBLIOGRAPHY

The following bibliography contains major references to recent research efforts in the assessment of individual and population exposure to environmental pollutants, and to the elucidation of exposure-dose relationships. The references are accompanied by brief abstracts.

# TOTAL EXPOSURE ANNOTATED BIBLIOGRAPHY

Aastrand, I. and F. Gamberale. Effects on Humans of Solvents in the Inspiratory Air: A Method for Estimation of Uptake. Environmental Research, 15(1):1-4, February 1978.

Forty-five male subjects aged 19-34 were exposed both at rest and during exercise to the following solvents: methylene chloride, trichloroethylene, styrene, and white spirit (83% aliphatic and 17% aromatic components). Total uptake of the solvents in the body was measured by the Douglas bag technique. Oxygen uptake, heart rate, arterial and venous blood concentrations, and alveolar air concentrations of the solvents were measured. The percentage uptake in the organism varied with the quotient of the concentrations in alveolar and inspiratory air. Body gas uptake approached zero as the alveolar concentration approached the inspiratory air concentration. The amount of a substance taken up may be estimated from the concentrations in the alveolar and inspiratory air and the pulmonary ventilation. This relationship is not valid for solvents easily soluble in water. In a number of psychomotor and cognitive tests administered during exposure to solvents, there was a clear relationship between impairment in performance and inspiratory air concentration of the solvent.

Ahuja, D., Kline, R. V., and W. Turner. Dynamic Modeling of Indoor Air Quality Using High Resolution Measurements. Presented at the International Symposium on Indoor Air Pollution, Health and Energy Conservation, John F. Kennedy School of Government, Harvard University, Amherst, Massachusetts, October 13-16, 1981. Abstract of Presentation.

Allen, R. J., R. A. Wadden, E. D. Ross. Characterization of Potential Indoor Sources of Ozone. American Industrial Hygiene Association Journal, 39(6):466-471, June 1978.

Ozone emission factors were developed for domestic electrostatic air cleaners and photocopying machines. The air cleaners had emission rates from 0-546 µg/min. These rates ordinarily will not produce air concentrations > 60 $\mu g/m^3$ (0.030 ppm) above background. The emission rate for photocopying machines varied from 48-158 µg/copy. In small poorly ventilated rooms, these emission rates were sufficient to produce incremental steady-state ozone concentrations of 396 $\mu g/m^3$ (0.202 ppm). Recent copying machine maintenance reduced ozone production to < 4 $\mu g/m^3$ (0.002 ppm), the detectable level.

Alzona, J., B. L. Cohen, H. N. Rudolph et al. Indoor-Outdoor Relationships for Airborne Particulate Matter of Outdoor Origin. *Atmospheric Environment*, 13(1):55-60, 1979.

The air in a room was cleaned, and airborne particulate matter was simultaneously collected for various time intervals in the room and outdoors nearby by pumping air through filters. The filters were analyzed by X-ray excitation for elements known to be primarily of outdoor origin (Fe, Zn, Pb, Br, and Ca). Within several hours an equilibrium is reached in which the indoor-outdoor ratio is typically 0.3. A model is developed incorporating filtration through walls, deposition on walls, and resuspension from surfaces in the room. Experiments were carried out with windows "cracked" open and wide open, and with windows and other room surfaces covered with plastic sheet to determine the importance of various terms. Several rooms of various types and two automobiles were studied. A person remaining indoors with doors and windows closed probably inhales no more than 1/3 as much dust of outdoor origin as he would if he were outdoors.

Anderson, I., G. R. Lundquist, and L. Molhave. Indoor Air Pollution Due to Chipboard Used as Construction Material. *Atmospheric Environment*, 9(12):1121-1127, 1975.

Measurements in 25 rooms in 23 dwellings in Denmark where particle board is used in walls, floors, and ceilings indicate that the average concentration is 0.62 and the range is 0.08-2.24 mg formaldehyde per cubic meter of air, exceeding the West German TLV for occupational exposure. A mathematical model for the room air concentration of formaldehyde is developed for predicting concentrations in rooms containing particleboard.

Ashford, N.A., D. Hattis, F.R. Heaton, J.I. Katz, W.C. Priest, and E.M. Zolt. Evaluating Chemical Regulations: Trade-off Analysis and Impact Assessment for Environmental Decision-Making. Center for Policy Alternatives, Massachusetts Institute of Technology, Cambridge, Mass, 1980.

Methods are discussed for analyzing the impacts of regulatory policy on human health. The implication of alternative dose-response and exposure-dose relationships are discussed. Case studies of a several federal regulations are used to illustrate the principles discussed.

Atkins, M.H., J. Lowe, and D. Lewis. Air Pollution and Human Health: A Case Study of Environmental Benefit Estimation. *International Journal of Environmental Studies*, 16(1):29-34, 1980.

Approaches to measuring the impact of air pollution on human health are an important aspect of environmental benefit estimation. Existing U.K. and U.S. evidence is reviewed, and some new evidence based on a cross

sectional study in the U.K. is presented. This is based on the premise that the effects of air pollution should be evaluated not on directly caused deaths but on their general impact on the environment which may act synergistically with other factors to affect longevity.

Babcock, L. R. and N. L. Nagda. (University of Illinois Medical Center) POPEX--Ranking Air Pollution Sources by Population Exposure. EPA-600/2-76-063. U.S. Environmental Protection Agency, Office of Research and Development, Washington, D. C., 1976. 345 pp.

The report gives results of research to develop quantitative models for relating emissions of air pollutants to their effects on people, and to use the methodology for determining the relative importance of air pollution sources. The quantitative methodology for ranking the sources developed in this project includes consideration of the dispersion of air pollutants, exposure of people, and subsequent health effects. The computer model, called POPEX, consists of three submodels: dispersion, population, and health effects. The model was applied to sources of air pollution in the Chicago Air Quality Control Region. Results show that 17 of 227 categories of sources contribute nearly 80% of the total air-pollution/population-effect problem.

Bakir, F., et al. Methylmercury Poisoning in Iraq. Science, 181:230-41, 1973.

The history of a methylmercury poisoning epidemic in Iraq is discussed, along with the relationships between blood mercury, total body burden and the development of neurological symptoms.

Behar, J. V., E. A. Schuck, R. E. Stanley, and G. B. Morgan. Integrated Exposure Assessment Monitoring. Environmental Science and Technology, 13(1):34-39, January 1979.

The integrated exposure assessment monitoring (IEAM) approach to pollution control is designed to assure that monitoring measurements are directly related to critical receptors and critical sources. The systematic set of guidelines can be standardized for all types of pollutants, including the definition of the area under consideration; studies on the exposure route and the existing air, water, and land quality; the definition of meteorological and hydrogeological situations; and the establishment of adequate monitoring and quality assurance systems.

Bellin, P. and J. D. Spengler. Indoor and Outdoor Carbon Monoxide Measurements at an Airport. Air Pollution Control Association Journal, 30(4):392-394, April 1980.

The indoor and outdoor environment at Logan International Airport's North Terminal were monitored in December 1978. Indoor concentrations were

significantly lower than the outdoor concentrations. The correlation coefficient between paired indoor-outdoor values was only 0.46. When the indoor values were offset by 15 minutes to allow for penetration and mixing indoors, the correlation coefficient increased modestly to 0.6. Outdoor concentrations are apparently log normally distributed with a mean of 9.8 ppm and a geometric standard deviation of 1.68. Applying Larsen's averaging predicted technique, a maximum 8 hr concentration of 14.6 is predicted. No violation of OSHA's occupational standard of 50 ppm 8 hr average was observed. There is apparently no problem with CO exposures to the general public.

Benson, F. B., J. J. Henderson and D. E. Caldwell. Indoor-Outdoor Air Pollution Relationships: A Literature Review. Prepared for the National Environmental Research Center, Research Triangle Park, North Carolina, August 1972,

Binder, R. E., C. A. Mitchell, H. R. Hosein, and A. Bouhuys. Importance of the Indoor Environment in Air Pollution Exposure. Archives of Environmental Health, 31(6):277-279, November-December 1976.

Bishop, Y.M.M., B.G. Ferris, and F.E. Speizer. Problems in Relating Health Effects to Air Pollution. Alta, Utah, July 5-9, 1976. Society for Industrial and Applied Mathematics, pp. 215-228, Philadelphia, 1977.

Although we know that dramatic episodes of fog or smog have been associated with increased mortality, our knowledge of the health effects of long-term exposure to lower levels of atmospheric pollutants is limited. The six-city study is designed to monitor both exposure to pollutants and health, particularly lung function, in both adults and children over a period of years. This paper discusses the rationale of the study design and how anticipated problems will be handled. Examples are given of analytic methods for handling questionnaire data.

Booz-Allen and Hamilton. Comparison of Four Methodologies to Project Emissions for the St. Louis Metropolitan Area. EPA/450/3-75/074, U.S. Environmental Protection Agency, Office of Air and Waste Management, Office of Air Quality Planning and Standards, Research Triangle Park, North Carolina, October 1975. 46 pp.

The report describes a comparison of four alternate methodologies which were used to project air pollution emissions for the metropolitan area. Two of the four methodologies also forecast expected ambient air quality levels. The purpose of the study was to summarize the structure and nature of each projection methodology, to compare the numerical projection results, and to discuss the relative advantages of each methodology.

Borst, F. J., R. L. Roswell, R. I. Scherpelz, and G. R. Hoenes. Accidental Dose Evaluation--External, Internal and Cross-Organ Doses. Health Physics, 37(6): 807, December 1979. Abstract only.

An effort to quantify the early effects (mortality and morbidity) of inhaled radionuclides is being performed at the Pacific Northwest Laboratory for the NRC. A computer program that calculates doses from simultaneous external and internal radiation exposure is being written for evaluating early effects to a population from an accidental release of any number or combination of radionuclides. The program calculates air concentration of radionuclides, assuming a bivariate normal distribution atmospheric model in a semi-infinite cloud. The Task Group Lung Model is used for determining lung burden and ICRP transfer coefficients for calculating organ burdens for most radionuclides. The ICRP-II lung model is being used for those radionuclides that do not behave asparticulates. Given the concentration at some time post-exposure and the location of the individual, external dose is calculated. The internal dose, including the cross-organ contribution, is calculated from the organ burdens.

Bruckman, L. and R. A. Rubino. Monitored Asbestos Concentrations in Connecticut. Air Pollution Control Association Journal, 28(12):122-1226, December 1978.

An air asbestos survey was conducted between late 1974 and early 1977 to define the magnitude of the health hazard posed by airborne asbestos fibers in Connecticut prior to the promulgation of the state's proposed asbestos air quality standard (30 $ng/m^3$ or 30,000 total asbestos $fibers/m^3$, 30 day average). A newly developed low volume particulate sampler, which operates continuously for 30 days at an air sampling flow rate of 4 cubic feet/minute and is equipped with special membrane filters, was used to collect ambient TSP samples for subsequent chrysotile asbestos electron microscopic determination. Ambient chrysotile asbestos levels removed from asbestos emission sources in urban and rural locations were $< 10$ $ng/m^3$; asbestos concentrations $> 30$ $ng/m^3$ were measured near each of the industrial users of asbestos. Asbestos levels adjacent to the toll plazas were also elevated (in the 10-25 $ng/m^3$ range), implicating asbestos emissions from vehicle brake lining decomposition as a significant source of airborne asbestos fibers. Indoor air asbestos levels were $< 1$ $ng/m^3$, suggesting that the risk to public health associated with the deterioration of asbestos surface coatings applied indoors may not be as severe as previously thought.

Bruno, R. C. Sources of Indoor Radon in Houses. Presented at the International Symposium on Indoor Air Pollution, Health and Energy Conservation, John F. Kennedy School of Government, Harvard University, Amherst, Massachusetts, October 13-16, 1981. Abstract of Presentation.

Buchet, J. P., H. Roels, G. Hubermont, and R. Lauwerys. Placental Transfer of Lead, Mercury, Cadmium, and Carbon Monoxide in Women. Part II. Influence of Some Epidemiological Factors on the Frequency Distributions of the Biological Indices in Maternal and Umbilical Cord Blood. Environmental Research, 15(3):494-503, June 1978.

The influence of various epidemiological factors (smoking habits, residence, age, occupation, drinking habits, duration of pregnancy, number of previous pregnancies) in the exposure of 474 European pregnant women and their newborns to Pb, Hg, Cd, and CO was investigated. The association of smoking with a reduction of fetal weight was confirmed. A slight but statistically significant effect of environmental pollution by Pb on Pb uptake by the pregnant mothers and its transfer to their fetuses was demonstrated.

Budiansky, S. Indoor Air Pollution. Environmental Science and Technology, 14(9):1023-1027, September 1980.

Indoor concentrations of pollutants often equal or exceed outdoor concentrations, and outdoor concentrations may have little to do with the true exposures to pollution. Determining when, where, and how exposure occurs is the key to limiting exposure and to understanding the fundamental effects of pollution. Indoor pollutants of concern include $NO_2$ from gas cooking, respirable particles from smoking, formaldehyde from insulation, BaP from burning wood, and radioactive Rn gas from building materials.

Buncher, C. R. (University of Cincinnati Medical Center), Cincinnati Drinking Water--An Epidemiologic Study of Cancer Rates. Prepared for the City of Cincinnati Board of Health, November 1975. 123pp.

Butler, J. D., and P. Crossley. An Appraisal of Relative Airborne Sub-Urban Concentrations of Polycyclic Aromatic Hydrocarbons Monitored Indoors and Outdoors. Science of the Total Environment, 11(1):53-58, January 1979.

Particle-size distribution studies of PAH in city and suburban atmosphere indicate that these compounds are associated with particles having mass median equivalent diameter values of 0.5 m. Ambient airborne concentrations of pyrene, chrysene, BaP, benzo(e)pyrene, and coronene at suburban sites between 11 and 17 km from a city center are nearly similar inside and outside detached residential houses. At all sites, monitored chrysene appears in highest concentration in the range of 4-7 $ng/m^3$, followed by BaP and benzo(e)pyrene at concentrations between 2 and 4 $ng/m^3$, with coronene generally <1 $ng/m^3$. The lungs of suburban populations will be exposed to approximately 34 ng/BaP/day, roughly equivalent to the exposure of BaP to the lung when 1 cigarette is smoked per day.

Calabrese, E.J. Pollutants and High-Risk Groups, J. Wiley, New York, 1978.

Cohen, A. F. and B. L. Cohen. Protection From Being Indoors Against Inhalation of Suspended Particulate Matter of Outdoor Origin. Atmospheric Environment, 14(2):183-184, 1980.

A method previously described for estimating the protection from being indoors against inhalation of suspended particulate matter of outdoor origin was applied to a wide variety of building and room types. The protection factor (PF) is approximated by the ratio of outdoor/indoor normalized collected quantities of suspended particulate matter. The average PF was 4.5 for large particles and 2.2 for submicron particles. Generally, Fe and Ca are associated with large particles (5µm), while Pb and Br are associated with submicron particles.

Colwill, D. M. and A. J. Hickman. Exposure of Drivers to Carbon Monoxide. Journal of the Air Pollution Control Association, 30(12):1316-1319, December 1980.

Eleven new cars were driven around a 35 km route comprising heavily trafficked roads in and around London, and the concentrations of carbon monoxide inside and immediately outside the vehicles were continuously monitored. Average levels of CO between 12 and 60 parts per million were found inside the cars, and these levels were between 30 and 80% of the external concentrations. The internal levels varied according to external changes but the changes were greatly damped by the buffering effect of the ventilation system.

Colome, S.D., J.D. Spengler, and S. McCarthy. Characterization of Aerosols and Inorganic Gases in Indoor Environments. Symposium on Indoor Air Pollution, Health, and Energy Conservation, Amherst, MA, 1981.

Colome, S.D., S. McCarthy, and J.D. Spengler. Residential Indoor and Ambient Outdoor Comparison of Gaseous and Particulate Air Pollutants in Two Cities. 74th Annual Meeting of the Air Pollution Control Association, Philadelphia, PA, June 21-26, 1981.

The results from one year of air monitoring in ten homes for six trace elements, $SO_2$, $NO_2$, $SO_4$, and respirable size particles are reported for Steubenville, Ohio and Portage, Wisconsin. Indoor and outdoor mean levels for all ten air constituents are higher in the industrial city of Steubenville. Indoor levels are lower than outdoor for most air constituents in both cities with the exception that mean levels of $NO_2$, respirable size particles and chlorine in Portage are higher indoors, indicating residential sources of these constituents. The measured pollution difference for this sample of homes is smaller if the mean indoor, rather than outdoor concentrations are compared. These findings have implications for previous and future epidemiologic studies of the effect of air pollution on human health.

Cook, N.R. The Steubenville Alert Data: Using Random Effects Models to Measure Acute Effects of Air Pollution on Pulmonary Function. Presented at Environmetrics 81, Conference of the Society of Industrial and Applied Mathemetics, Alexandria, VA, April, 1981.

Cool, D. A., W. S. Cool, A. Brodsky, and G. G. Eadie. Estimation of Long-Term Biological Elimination of Insoluble Iradium-192 from the Human Lung. Health Physics, 36(5):629-632, May 1979.

In 1967, 2 individuals were involved in an inhalation accident as a result of a mistake in cutting into a 2,000 Ci source. About 14 hr. after exposure, measurements indicated a deposition of 412 muCi in the lungs of Subject 1 and 278 muCi for Subject 2. Data on the rate of clearance were obtained over a 2-yr. period. Urine and fecal samples were collected, and lung and whole body scans were performed. A single term exponential fits the retention data collected over the 2-yr. period. Differences were noted in the effective half-life values determined by the 2 detector systems used, possibly as a result of the biological translocation of the material over time. Data indicate that the particulate material moves from the pulmonary alveolar region to the trachiobronchial, bronchopulmonary, and medistinal lymph nodes. Lung clearance rates determined by analysis of fecal samples indicated a range of biological half-live values in the order of T 1/2 of 1,000.

Cortese, A.D. Ability of Fixed Air Monitoring Stations to Represent Personal Carbon Monoxide Exposures. Ph.D. Dissertation, Harvard University, School of Public Health, 1976.

Deinzer, M., F. Schaumburg, and E. Klein. Environmental Health Sciences Center Task Force Review on Halogenated Organics in Drinking Water. Environmental Health Perspectives, 24:209-239, June 1978.

Epidemiological studies suggested a higher incidence of cancer along the lower Mississippi River where the contamination from organic chemicals is particularly high. The task of assessing the effects of chemicals in the drinking water includes many variables, including differences in water supplies and the temporal relationship between contamination and consumption of the finished product. It must also take into account the relative importance of the effects of these chemicals in comparison to those of occupational exposure, ingestion of contaminated foods, inhalation of polluted air, and many others. The susceptibility of different age, genetic, and ethnic groups within the population must also be carefully considered. Discussed are the reasons for disinfection; the general occurrence of chlorinated organics in drinking water; the chemistry in the synthesis of chlorinated organics under aqueous conditions; and alternatives to chlorine for disinfection.

Dennis, R. L. An Air Pollution Dispersion Model for Long-Range Policy Analysis. Ecological Modeling, (2):121-141, March 1980.

The smeared concentration approximation (SCA) method of determining air pollution dispersion is described. The SCA method focuses on urban area analysis. Using this method, the impacts of air pollution dispersion can be included in multidisciplinary studies for long-term policy analysis. The SCA dispersion parameters are used to calculate a spatially averaged ground level exposure for each of three emmission classes. The SCA air pollution dispersion model is validated, and three examples of air pollution policy analysis based on case studies are presented.

DiVencenzo, G.D. and M.L. Hamilton. Fate of n-Butanol in Rats after Oral Administration and Its Uptake by Dogs after Inhalation or Skin Application. Toxicology and Applied Pharmacology, 48:317-325, 1979.

DiVincenzo, A.D., and C.J. Kaplan. Effect of Exercise and Smoking on the Uptake, Metabolism and Excretion of Methylene Chloride Vapor. Toxicology and Applied Pharmacology, 49:141-148, 1981.

The authors studied the effects of smoking and exercise on the intake of inhaled methylene chloride. They found that exercise increased the rate of uptake, metabolism, and excretion of methylene chloride, but did not increase the peak levels of carboxyhemoglobin in the blood.

Dockery, D.W., and J.D. Spengler. Indoor-Outdoor Relationships of Respirable Sulfates and Particulates. Atmospheric Environment, Vol. 15, pp. 335-343, 1981.

Indoor and outdoor concentrations of respirable particulates and sulfates have been measured in 68 homes in six cities for at least one year. A conservation of mass model was derived describing indoor concentrations in terms of outdooor concentrations, infiltration, and indoor sources. The measured data were analyzed to identify important building characteristics and to quantify their effect. The mean infiltration of outdoor fine particulates was found to be approximately 70 %. Cigarette smoking was found to be the dominant indoor source of respirable particulates. Increased indoor concentrations of sulfates were found to be associated with smoking and also with gas stoves. The effect of full air conditioning of the building was to reduce infiltration of outdoor fine particulates by about one half, while preventing dilution and purging of internally generated pollutants. The model for indoor respirable particulate and sulfate levels was found to compare well with measurements.

Dockery, D. W. and J. D. Spengler. Personal Exposure to Respirable Particulates and Sulfates. Air Pollution Control Association Journal, 31(2):153-159, February 1981.

As part of a long-term epidemiologic study of the respiratory effects of air pollution, a monitoring program to measure personal exposure to respirable particulates and sulfates was conducted. The personal exposure of 37 volunteers to these substances was measured and compared with simultaneous measurements of outdoor ambient concentrations and concentrations in the homes of the participants. Results indicated that mean personal exposure was directly related to mean outdoor concentrations of particulates and sulfates. A time-weighted indoor/outdoor model to estimate personal exposure to respirable sulfates and particulates is described.

Dockery, D.W., J.D. Spengler, M.P. Reed, and J.H. Ware. Relationships Among Personal, Indoor, and Outdoor $NO_2$ Measurements. Presented at the 73rd Annual Meeting of the Air Pollution Control Association, Montreal, Canada, June 23-27, 1980. Submitted to Environment International.

Using integrating $NO_2$ diffusion dosimeters, personal, indoor and outdoor exposures were measured for nine families in Topeka, Kansas. $NO_2$ exposures in homes that used gas for cooking were clearly different from those in homes that used electricity. The gas-cooking homes had indoor levels three times the outdoor levels. Members of the gas-cooking households had levels twice those of electric-cooking families and twice the outdoor levels. A linear model that includes outdoor concentrations and stove types explains 77% of the variance in observed in $NO_2$ exposure. The differential exposures in homes with and without gas stoves should be considered in epidemiologic studies of the health effects of air pollution.

Drake, R. L. Battelle Pacific Northwest Laboratory). Interfacing Air Pathway Models with Other Media Models for Impact Assessment. Presented at the 20th Hanford Life Sciences Symposium, Richland, Washington, October 19-23, 1980. U.S. Department of Energy Contract No. DE-AC06-76RLO-1830, October 1980.

Eifler, C. W., S. S. Stinnett, and P. A. Buffler. Simple Models for Community Exposure from Point Sources of Pollution. Environmental Research, 25(1):139- 146, June 1981.

Several simplyfying steps have been proposed for the Gaussian Plume Model for application to community exposure from point sources of pollution investigations. These steps lead to exposure indicator functions requiring far less data gathering and computation, but introduce some errors. The magnitude of these errors is examined. Research is underway to examine the utility of such models in studies of respiratory and other cancers in highly industrialized communities near chemical or petrochemical plants.

Environmental Health Resource Center. A Model for Establishing an Ambient Air Standard for Mercury in the State of Illinois. IIEQ No. 76/0, Illinois Institute for Environmental Quality, Chicago, Illinois, October 1976. 29 pp.

This paper presents a method for the establishment of a mercury body burden based on neurological toxic symptoms. Among the topics covered are the following: toxic effects at low levels of mercury exposure (human chromosomal aberrations, further effects of mercury on humans); determination of safe maximum daily intake of mercury for males and females; daily dietary intake of mercury for humans (intake of mercury via fish, mercury intake from food other than fish, intake of mercury via water); establishment of an ambient air standard for mercury (calculation of mercury standard, why the mercury standard is based on a two week period); determination of percent of population protected under different air standards; toxicity; and safety factors.

Ferrari, R. L. and D. H. Jones. An Upper Limit Analysis of Carbon Monoxide Monitoring in the United States. Air Pollution Control Association Journal, 29(10):1062-1063, October 1979.

The most practical means to determine whether available monitoring data are representative of the highest ambient 8-hr average CO levels experienced in major cities is to inventory the critical characteristics of monitor siting. Such data were catalogued for the worst station in 8 to 12 counties with the highest number of CO violations in the nation. The local agencies for the 8 counties with 90 annual violations provided site configuration data for the worst station in their county. Data for Fairbanks, Chicago, Morristown, Spokane, Cleveland, Albuquerque, Louisville, and Denver are tabulated. Probes at all 8 stations were located at heights of 2.4-7.0 m. Actual traffic counts of the adjacent roadways were available for 1/2 the sites which were close to or actually met the EPA recommended criteria of 10,000 vehicles/day. The highest and lowest maximum 8-hr observation values are presented graphically.

Ferris, B.G., Jr., F.E. Speizer, Y.M.M. Bishop, J.D. Spengler, and J.H. Ware. The Six City Study: A Progress Report. Atmospheric Sulfur Deposition: Environmental Impact and Health Effects, Edited by Shriner, Richmond, and Lindberg. Ann Arbor Science Publishers, Inc., Ann Arbor, MI. pp. 99-108, 1980.

The Six-City Study was designed to test the adequacy of the present primary standards for $SO_2$ and particulates and whether they should be relaxed or made more stringent. The study was originally designed to examine the chronic effects of air pollutants. To accomplish this two relatively clean cities were selected, two with levels slightly below the primary standards, and two dirty cities that had levels often above the standards. The cities are: clean-Topeka, Kansas, and Portage, Wisconsin; moderate-Watertown, Massachusetts, and Kingston-Harriman, Tennessee; and dirty-Steubenville, Ohio, and the southern tip of St. Louis, Missouri.

Fischbein, A., A. N. Rohl, A. M. Langer, and I. J. Selikoff. Drywall Construction and Asbestos Exposure. American Industrial Hygiene Association Journal, 40(5):402-407, May 1979.

The rapid development of the drywall construction trade in the U.S. is described. It is estimated that some 75,000 U.S. construction workers are currently employed in this trade. The use of a variety of spackle and taping compounds is associated with significant asbestos exposure; air samples taken in the breathing zone of drywall tapers during sanding of taping compounds show fiber concentrations exceeding the maximum level permitted by federal regulations. The results of a clinical field survey of drywall construction workers demonstrating that asbestos disease may be an important health hazard are presented.

Fischbein, L. Potential Halogenated Industrial Carcinogenic and Mutagenic Chemicals: IV. Halogenated Aryl Derivatives. Science of the Total Environment, 11(3):259-278, April 1979.

Ortho- and para-dichlorobenzenes, 1,2,4-trichlorobenzene, HCB, bromobenzenes, and benzylchloride were reviewed principally in terms of their synthesis, areas of utility, stability, distribution, reactivity, levels of exposure, populations at risk, metabolism, carcinogencity, and mutagenicity.

Fiserova-Bergerova, V. Predicting "Individual Differences" in Uptake and Excretion of Gases and Lipid Soluble Vapors: Simulation Study. British Journal of Industrial Medicine, 31:42-49, 1974.

This paper lays out the theoretical basis for the development of a pharmacokinetic model for the uptake and metabolism of organic vapors in the body. An analogy is made to principles of electrical circuitry with the affinity of organs and transport rates being simulated by electrical resistors and capacitors.

Fiserova-Bergerova, V. Predicting "Individual Differences" in Uptake and Excretion of Gases and Lipid Soluble Vapors: Simulation Study. British Journal of Industrial Medicine, 37:42-49, 1980.

The fully-developed pharmacokinetic model previously described (Fiserova-Bergerova, 1974) is used to examine differences in the uptake and metabolism of organic vapors that can be expected to be a result of physiological differences (e.g., body build) between individuals.

Fiserova-Bergerova, V. Modeling of the Uptake and Clearance of Inhaled Vapors and Gases. Industrial and Environmental Xenobiotics, I. Gut, et al., editors, Springer-Verlag, NY, 1981.

The basic pharmacokinetic model (Fiserova-Bergerova, 1974) and some of its predictions concerning individual differences in pollutant uptake are reviewed. The implications of Michaelis-Mentin kinetics for uptake and metabolism are described.

Gardner, D. E., D. L. Coffin, M. A. Pinigin, and G. I. Sidorenko. Role of Time as a Factor in the Toxicity of Chemical Compounds in Intermittent and Continuous Exposures, Part 1--Effects of Continuous Exposure. Journal of Toxicology and Environmental Health, 3(5-6):811-820, December 1977.

Gasper, J. R. and P. A. Dauzvardis. (Argonne National Laboratory, Argonne, Illinois) Body Burdens--An Integrated Approach to Health Risk Assessment. Prepared for 1979 National Conference on Hazardous Material Risk Assessment, Disposal and Management, Miami Beach, Florida, April 25-27, 1979. 18 pp.

Comparison of body burdens and critical organ burdens can identify the relative contribution from different sources of exposure, exposure pathways, and routes of entry. These two burdens may be used in conjunction with monitoring data and environmental modeling techniques to assess the relative risk of effluent releases from natural and industrial sources or from different effluent control technologies or waste disposal methods. The relative impact of different levels of regional economic growth or regulatory plans can also be assessed. Body burdens may also serve to indicate where effluent control efforts will be most successful in reducing impacts on humans.

Good, B. W., G. Vilcins, et al. Effect of Cigarette Smoking on Residential $NO_2$ Levels. Environment International, 8:167-183, 1982.

Hales, C. H., A. M. Rollinson, and F. H. Shair. Experimental Verification of Linear Combination Model for Relating Indoor-Outdoor Pollutant Concentrations. Environmental Science and Technology, 8(5):452-453, May 1974.

The linear combination model appears to provide an "upper bound" in relating indoor concentrations of ozone to those outside. Tests conducted with a rather large office laboratory indicated that the model is in excellent agreement with ozone concentrations in hallways. The model is in good agreement with ozone concentrations in rooms; however, ozone concentrations vary somewhat from room to room depending on the local ventilation and geometric parameters.

Harley, J. H. Radon and Radon Daughter Dose Implications. Health Physics, 37(6):818-819, December 1979. Abstract only.

The lung dose from Rn daughters is the highest exposure received by man from the natural environment. The enhanced levels produced by man's activities as in Grand Junction and Canada may be several times higher than the natural levels. Widespread measurements are being made of indoor concentrations under natural conditions as a true basis for comparison with enhanced levels. The physical dosimetry appears to be setting down to reasonable agreement, but the conversion of dose to health effects is still the subject of argument.

Hedden, K.F. Water Treatment Model for Pollutant Exposure Assessment. Journal of Environmental Science and Health-Environmental Science and Engineering, A15(4):285-305, 1980.

A mathematical model of potable water treatment for toxic substance removal has been developed for use in exposure assessment studies. Various treatment processes are incorporated into the model. These include sedimentation, coagulation, flocculation, filtration, aeration, chemical oxidation, activated carbon adsorption, and chlorination. This model provides a means of predicting the quantity of toxic substances in tap water following treatment.

Hegde, A. G., D. M. Thakker, and I. S. Bhat. Long-Term Clearance of Inhaled 60Co. Health Physics, 36(6):732-734, June 1979.

Five nuclear power station workers exposed to Cobalt-60 through inhalation were monitored for clearance of lung-deposited activity for periods ranging from 120-450 days. A shadow-shield whole-body monitor was used for measuring the Co, and care was taken to prevent fresh exposure during the study. The cases included one acute single exposure, 2 multiple exposures, and 2 chronic exposure cases. Cobalt-60 activity was confined to the chest, and the average biological half-life observed was 664 days, which agrees closely with the ICRP values.

Hertz, M. B., Mage, D. B., and Heiderscheit, L. T. (The Research Corporation) Development of Methodology to Improve the Characterization of Human Exposure in CHAMP Study Areas. Presented at the 71st Annual Meeting of the Air Pollution Control Association, Houston, Texas, June 25-30, 1978. 12 pp.

The Acute Respiratory Disease Study of the Population Studies Division of the Health Effects Research Laboratory is designed to measure the acute response in respiratory disease from peak hourly and daily average exposure to nitrogen dioxide ($NO_2$) alone and in combination with other pollutants (especially $O_3$). Pollutants, other than $NO_2$ and $O_3$, that will be considered are total suspended particulates, respirable suspended particulates, total hydrocarbons, non-methane hydrocarbons, carbon monoxides, sulfur dioxide, suspended sulfates, and suspended nitrates. Presently, a single station in each study community is being used as the estimate of exposure for each study participant in that area. The approach of the study is to define as accurately as possible, based on the normal mobility patterns of the study population, the major components of pollutant exposure; to develop relationships to allow exposure to be estimated from identifiable variables; to evaluate the validity of these relationships; and to identify and quantify the sources of error in these relationships.

Hewett, P. J. Inhalation of Dusts. Journal of Environmental Management, 8(1):9-12, January 1979.

Methods used to establish quantitatively the exposure of the respiratory tract, including lungs to dusts are discussed. To enter the respiratory tract, dust particles must be airborne and be sufficiently light to be drawn in during inspiration. The relationship between environmental dust concentration and amount and site of actual deposition in the worker can be established if the particle size is known. Dusts 5 micrometers in diameter are considered respirable. Static samples positioned in selected areas of the workplace, while conveniently used for dust collection, are not as valuable as personal samplers operating in the workers' breathing zone, which allow dust to be collected, weighed, and analyzed for chemical constituents, and the particle size determined. Means of establishing the site of initial deposition of dust and the amount deposited are discussed. Assessment of the dose to a particular part of the respiratory tract must consider the amount initially deposited, the mucociliary clearance rate, and the amount retained following exposure. More extensive use of the personal monitor will indicate a need for modification to the workplace or redeployment of the worker based on his dose record, and the record can be related to the health of groups of workers, aiding epidemiological studies.

Hickey, J. L. S. and P. C. Reist. Adjusting Occupational Exposure Limits for Moonlighting, Overtime, and Environmental Exposures. American Industrial Hygiene Association Journal, 40(8):727-733, 1979

A mathematical model predicts relative body burdens of inhaled contaminants in workers who work two jobs or overtime, or who experience off-the-job exposure to air contaminants. Expected "peak" or maximum body burdens from multiple exposures are compared to those expected from the "normal" occupational exposure on which TLVs and Permissible Exposure Limits (PELs) are based (five 8-hr d/wk with zero off-the-job exposure). The model predicts what adjustments to the TLV and PELs are necessary to avoid accumulation of excess peak body burdens of contaminants from the additional exposures incurred. The model takes biological uptake and excretion directly into account using first-order kinetics. The general application of models to determine occupational exposure limits is reviewed, and several models are compared.

Hoggan, M. C., A. Davidson, et al. Motor Vehicle Emissions and Atmospheric Lead Concentrations in the Los Angeles Area. Journal of the Air Pollution Control Association, 28(12):1200-1206, December 1978.

This paper is directed to those concerned with the effect of changes in lead consumption by motor vehicles on atmospheric lead concentrations. Atmospheric lead concentrations in the Los Angeles area have been found to be dependent on lead consumption, meteorology, and source-receptor relationships. Mathematical relationships between these variables for selected Los Angeles area sites are derived. Los Angeles County experienced a 50 percent reduction in annual average atmospheric lead concentration between 1971 and 1976, which was found to be due to a decrease in vehicular emissions of lead. Seasonal variations in lead of concentration were found to be mainly a product of seasonal variations in weather. Projections of atmospheric lead concentrations for the next decade show a continued downtrend, based on regulation of fuel lead content and introduction of additional catalyst equipped vehicles. By the mid-1980's most locations in the Los Angeles area are expected to attain the California and federal ambient air quality standards for lead of 1.5 micrograms per cubic meter, based on a monthly average.

Hollowell, C. D. Measurements of Air Quality in the Bay Area: Outdoor and Indoor. American Physical Society, 23(1):41, January 1978. (abstract only)

Major programs by several groups are under way to characterize air pollutants in the San Francisco air basin. Such characterization depends on measurement of the chemical and physical properties of pollutants and identification of the transformations which they undergo. Work at the Lawrence Berkeley Laboratory concentrates largely on the outdoor urban air of the Bay area, but also includes studies of indoor air quality. A major goal of the urban field characterization study is to establish what differences, if any, can be found in the chemical and physical composition of winter and summer air pollution, when the photochemical activity is, respectively, low and high. An important facility in this work is the Mobile Atmospheric Research Laboratory, a van containing a comprehensive array of monitoring instrumentation. This instrumentation has also been used for measurements in private homes as a basis for understanding the possible impact of indoor air pollution on human health. Overall, these measurement efforts are focused on better characterization of population exposures to air pollution in all environments, outdoor and indoor.

Hollowell, C. D., Berk, J. V., and G. W. Traynor. Indoor Air Quality Measurements in Energy Efficient Buildings. Presented at the 71st Annual Conference of the Air Pollution Control Association, Houston, Texas, June 25-30, 1978. 16 pp.

The monitoring program includes measurement of carbon monoxide, carbon dioxide, sulfur dioxide, nitrogen oxide, nitrogen dioxide, ozone, infiltration rate (tracer gas technique), and aerosol size distribution on a continuous basis. Total and respirable-fraction particulate samples are collected on membrane filter media for analysis by X-ray fluorescence, photoelectron spectroscopy, proton activation analysis, combustion, and wet-chemistry techniques for the determination of particulate elemental composition (S, N, C, etc.) and ionic species such as sulfate, nitrate, and ammonium. Results of the initial phases of this program indicate that the concentrations of some gaseous and respirable particulate air pollutants in specific indoor environments exceed those levels commonly found in the outdoor urban air environment.

Holma, B., G. Kjaer, and J. Stokholm. Air Pollution, Hygiene and Health of Danish School Children. *Science of the Total Environment*, 12(3):251-286, August 1979.

A comprehensive investigation forming part of a joint European study under the auspices of "WHO Long-Term Air Pollution Programme" used 7-13 yr-old schoolchildren as the target group. The study included social, housing, hygienic, and epidemic factors as well as family smoking habits. At exposure to low levels of air pollution, these factors dominate as causes for the impairment of health, especially that of respiratory health.

Ishizu, Y. General Equation for the Estimation of Indoor Pollution. *Environmental Science and Technology*, 14(10):1254-1257, October 1980.

Several general equations are developed, 2 of which are appropriate to estimate the concentration of indoor pollution. In experiments performed to determine smoke generation rates, filter tip efficiency, and mixing factors, various numbers of cigarettes were smoldered, or smoked on a standard smoking machine, in different sized rooms, with and without ventilation at known rates. When the mixing factor (usually defined as the ratio of the effective air changes to the theoretical air changes) was unity, experimental results did not agree with calculated ones. When it was applied following developed formulas, calculated values agreed closely (using the developed equations), when the mixing factor was 0.4. It was noted that when a value of the volume rate of indoor intake (for ventilation), divided by the volume of the room, becomes larger, the mixing factor becomes smaller.

Jabara, J.W., *et al*. Carbon Monoxide: Dosimetry in Occupational Exposures in Denver, Colorado. *Archives of Environmental Health*, 35:198-206, 1980.

Johansson, I. Determination of Organic Compounds in Indoor Air with Potential Reference to Air Quality. *Atmospheric Environment*, 12(6-7):1371-1377, 1978.

Concentrations of 15 volatile organic compounds were investigated in the air of two Swedish schoolrooms. The chemical analysis included enrichment on porous polymer beads, heat desorption, and GC separation on a capillary column connected to either a flame ionization detector or an MS. Samples were collected from the indoor air both in the presence and in the absence of the pupils as well as from the ambient outdoor air. The qualitative composition of indoor and outdoor air was about the same; aliphatic and aromatic hydrocarbons predominate, though indoors the number of compounds detected is larger and the concentrations are higher. Both the number and the concentration increase in the presence of humans. The mean concentrations of acetone and the sum of the concentrations of C2-alkylbenzene were 7.7 and 8.2 $\mu g/m^3$, respectively, in an unoccupied room; these increased to 19.8 and 12.1 $\mu g/m^3$, respectively, in an occupied room.

Joumard, R., et al., Mathematical Models of the Uptake of Carbon Monoxide on Hemoglobin at Low Carbon Monoxide Levels. Environmental Health Perspectives, 41:277-290, 1981.

A set of equations is developed for relating carboxyhemoglobin levels to carbon monoxide exposures over time. The equations are used to assess carboxyhemoglobin levels over the course of a week, using a simple model of receptor behavior.

Kanarek, H. C., K. J. Yost, and V. L. Anderson. A Study of Cancer Mortality in an Urban Industrial Environment. Journal of Environmental Science and Health--Part A: Environmental Science and Engineering, A14(8):641-681, 1979.

Recent literature is reviewed, suggesting a relationship between urban air pollution and cancer incidence. The areas investigated include a highly urbanized, heavily industrialized region and a rural, relatively unpolluted area in northwestern Indiana. The data base for the study included mortality data, population statistics, and air pollution inventory. TSP data were used to estimate the respiratory exposure of the study population to airborne carcinogens, and a computer program was developed to extend the data to a population within a geographic area. A second program computed cancer mortality rates within a geographic area. Data shows that the mortality rate for malignant neoplasm of the treachea, bronchus, and lung increases in the low pollution group with time, possibly due to a migration from urban to rural areas which occurred during the time frame investigated. For all categories of malignant neoplasm the cancer mortality rates are generally higher in the urban area than in the rural area.

Kershaw, T.G. The Relationship Between Blood Levels and Dose of Methyl Mercury in Man. Archives of Environmental Health, 35:28-36, 1980.

The relationship between methylmercury intake, blood levels and adverse effects is discussed. The author points out how short-term, high mercury exposures (from eating contaminated fish, for example), may produce spuriously high blood mercury levels which do not reflect recent average exposure levels.

Kjellstrom, T. and Nordberg, G. A Kinetic Model of Cadmium Metabolism in the Human Being. Environmental Research, 16:248-269, 1978.

A multi compartment pharmacokinetic model is presented and used to analyze the relationship between cadmium intake for various sources and levels in the kidney, a major target organ.

Knelsen, J.H. Problems of Estimating Respiratory Lead Dose in Children. Environmental Health Perspectives, 35:53-58, 1974.

The problems associated with estimating dose in children are discussed. Most important among these is children's elevated respiratory rate, which is approximately twice as high for children as for for adults. Other sources of uncertainty in assessing children's lead dosages are discussed.

Konz, J. and P. Walker. An Assessment of Cadmium in Drinking Water from a Multi-Media Perspective. The MITRE Corporation, U.S. EPA Contract No. 68-01-4635, September, 1979.

Krisiuk, E. M. Airborne Radioactivity in Buildings. Health Physics, 38(2):199-202, February 1980.

Expressions for the concentration of 220 Rn and 222 Rn daughters in indoor air have been derived. Taking into account the exhalation rate and diffusion processes of building materials, room dimensions, and ventilation rates, the mean radon and thoron daughter concentrations in indoor air are estimated at 0.10 pCi/l and 0.0064 pCi/l, respectively. These values correspond to a lung dose equal to 126 mrem/yr.

Kusuda, T., S. Silberstein, and P. E. McNeil, Jr. Modeling of Radon and its Daughter Concentrations in Ventilated Spaces. Air Pollution Control Association Journal, 30(11):1201-1207, November 1980.

To predict indoor radiation levels due to Rn daughters at low building ventilation and air leakage rates, differential equations governing the decay and venting of 222 Rn and its daughters were used. A computer program based on the equations was written to predict 222 Rn and daughter concentrations, total potential alpha energy concentration, and equilibrium factor. The program can account for time dependence of ventilation and emanation rates and is readily used by building designers. Sample calculations using the program showed that potential energy levels in tightened buildings can commonly reach 0.01 working level, a level more than 2 times as high as concentrations currently found in most houses.

Kutz, F. W., R. S. Murphy and S. C. Strassman. Survey of Pesticide Residues and Their Metabolites in Urine from the General Population. Reprinted from "Pentachlorophenol" (1978), Edited by K. Ranga Rao, Plenum Publishing Company, New York.

The National Human Monitoring Program for Pesticides is collaborating with the U.S. Public Health Service in a three year study to assess the exposure of the general population of the United States through analysis of human urine for residues of selected pesticides and their specific metabolites. Samples are collected on a national probability design and analyzed for selected organochlorine, carbamate, chlorophenoxy and organophosphorous pesticides. Medical, nutritional and pesticide usage data are available from each donor.

Kutz, F. W., A. R. Yobs and H. S. C. Yang. National Pesticide Monitoring Programs. Reprinted from "Air Pollution from Pesticides and Agricultural Processes. Robert E. Lee, Jr., (ed.), Cleveland, Ohio: CRC Press, Inc.

Kutz, F. W., S. C. Strassman, A. R. Yobbs, and J. F. Viar, Jr. Pesticides in People. Reprinted from Pesticides Monitoring Journal, II(1):61-63, September 1977.

Agricultural uses of the insecticide DDT were cancelled by the U.S. Environmental Protection Agency on December 31, 1972. However, the domestic use of DDT had begun to decline before this action. Beginning July 1969, residues of DDT and its metabolites were measured in human adipose tissue collected through an annual national survey. Levels of total DDT equivalent residues in human adipose tissue have decreased slightly, but the frequencies of finding DDT or its metabolites have remained high. The most marked decline in residue concentration has been found in the youngest age group 10-14 years). Approximately 80 percent of the total DDT equivalent found in this survey was DDe. These data show that the reduction of the agricultural uses of DDT has decreased human exposure to and storage of this chemical.

Kutz, F. W.,, S. C. Strassman and J. F. Sperling. Survey of Selected Inorganic Pesticides in the General Population of the United States. Reprinted from the Annals of the New York Academy of Sciences, Volume 320, pp. 60-68, May 31, 1978.

In decisions regarding the uses of aldrin, dieldrin, heptachlor, and chlordane, the Administrator of the U.S. Environmental Protection Agency cited and considered the findings of these chemicals in human tissues. Findings of pesticide residues and their metabolites in representative samples from the general population provide a major element in pesticide regulatory decision-making. These residues demonstrate the extent of the environmental distribution of particular pesticides and, when coupled with laboratory animal data showing adverse biologic effects, signal a potential public health hazard.

One program that examines pesticide residues and associated chemicals in human tissues and fluids is the National Human Monitoring Program for Pesticides. This program operated by the U.S. Environmental Protection Agency, monitors on a national scale, the prevalence and level of exposure to selected pesticides experienced by the general population. These data are used to identify trends in this exposure and to assess the effects of regulatory actions.

This paper reports findings of selected organochlorine pesticide residues and their metabolites in human adipose tissue collected annually on a national basis from fiscal years 1970 through 1975.

Lamouraeux, R. L., K. Hirschinger and D. H. Kearin. (System Development Corp.) A Survey of Average Driving Patterns in the Houston Urban Area. Under contract no. PH 22-68-56 with the National Center for Air Pollution Control, Department of Health, Eduction and Welfare, and the Coordinating Research Council, Inc. August 29, 1969. 95 pp.

Landau, E. and N. Manos. Epidemiology Studies--Estimation of Population Residing Near Vinylidene Chloride Plants. EPA/560/6/76-022, U.S. Environmental Protection Agency, Office of Toxic Substances, Washington, D.C., August 1976. 121 pp.

The report presents estimates of the population residing within five miles of plants currently producing or processing vinylidene chloride and the methodology used for estimation. It is estimated that 3.6 million persons reside within five miles of the 38 plants that have been identified. These estimates are presented by age, sex, direction and distance from the plant for each of the 38 plants individually. For the plants located in Standard Metropolitan Statistical Areas that have been divided into census tracts, the population was estimated from Bureau of the Census publications given the 1970 population for census tracts. For plants located in untracted areas, the estimates were made using Bureau of the Census maps of county subdivisions and publications giving the 1970 population for county subdivisions.

Leaderer, B. P. Estimates of Health Benefits Due to Reductions in Ambient NO2 Levels. Environmental Management, 1(1):31-37, 1976.

A method is developed to estimate the actual number of individuals in the U.S. expected to suffer respiratory illness from exposure to ambient nitrogen dioxide concentrations exceeding the federal standard. Approximately $33.2 \times 10^6$ excess cases of respiratory illnesses in 1973 were associated with $NO_2$. A reduction of 50-60% below 1973 $NO_2$ levels is needed to eliminate excess respiratory illness associated with the ambient $NO_2$ concentration.

Lioy, P. J., J. G. Watson, Jr., and J. D. Spengler. APCA Specialty Conference Workshop on Baseline Data for Inhalable Particulate Matter. Air Pollution Control Association Journal, 30(10):1126-1130, October 1980.

The workshop was held to identify existing data on the size distributions and mass concentrations of the atmospheric aerosol and the general features of these data applicable to the characterization of size fractionated aerosol contributions under average and episodic conditions. The hypotheses formulated were as follows: there are no consistent or constant relationships among EPA inhalable particulate matter (IPM), total suspended particulate matter (TSPM), and British Smoke (BS); in the U.S., the fine particle concentration of the atmospheric aerosol has not increased over the last 70 yrs; 50% of the fine particle (FP) mass concentration is of anthropogenic origin; it is not possible to determine the transport of the fine particles into an airshed; and the relationship between EPA-IPM and TSPM can be defined with specific categories characterized by emissions sources, meteorological conditions, and site location. The hypotheses may or may not be completely answerable within the framework of existing data bases on the atmospheric aerosol. A list of major studies conducted in the U.S. and one from Canada is presented to provide a reference point for discussion of exposure to IPM-EPA and FP. Identifying information is provided, with emphasis on location and protocols for the study, size ranges sampled, and chemical analyses.

Mahaffey, K. R. and J. E. Vanderveen. Nutrient-Toxicant Interactions: Susceptible Populations. Environmental Health Perspectives, 29:81-87, April 1979.

Nutritional status can substantially modify the toxicity of environmental pollutants. Investigations with experimental animals and epidemiological observations on humans established the role of nutrition in altering susceptibility to a variety of pollutants including pesticides and heavy metals. Adsorption of the toxicant from the gastrointestinal tract may be influenced by the dietary intake of nutritionally significant compounds. Nutrients also can influence cellular toxicity by sequestering the toxic compound in adipose tissue or bone mineral where it is relatively unavailable. Changes in diet can produce alterations in the concentrations of key nutrients involved in detoxification reactions. The degree of nutritional deficiency that alters susceptibility need not be severe. Frequently only biochemical indications of nutritional deficiency can be associated with change in the dose-response of an animal or person to a toxic compound. Feeding higher levels of nutritionally significant compounds does not provide protection against toxic compounds. Understanding the physiological basis of nutrient-toxicant interactions is a key to identifying practical uses of nutrition in assessing population groups at risk and devising dietary strategies to minimize adverse effects of exposure to environmental toxicants. Although diets and environments of experimental animals can be manipulated to create nutrient deficiencies, the likelihood of some deficiencies occurring spontaneously in a human population may be very small.

Marcus, A.H. The Body Burden of Lead: Comparison of Mathematical Models. Environmental Research, 19:79-90, 1979.

The author compares two recently proposed pharmacokinetic models for lead metabolism in humans and investigates the effects of changes in model parameter values and structure on predictions about body burden as a function of exposure over time. An alternative model is advanced which seems to predict long-term lead uptake more accurately than the other models discussed.

McGregor, R. G. and L. A. Gourgon. Radon and Radon Daughters in Homes Utilizing Deep Well Water Supplies, Halifax County, Nova Scotia. Journal of Environmental Science and Health--Part A: Environmental Science and Engineering, A15(1):25-35, 1980.

The concentration-time of radon in tap water supplies varied from 43 nCi/l to 370 nCi/l. Indoor air concentrations varied from 0.5 pCi/l to 19.1 pCi/l and 0.001 WL to 0.025 WL for radon and radon daughters, respectively. The transfer of radon from tap water raised the concentration in the bathroom air during operation of the shower. Transfer efficiencies of 37-70 percent were observed.

McKenzie, R. L., D. S. Bright, et al. Development and Characterization of Personal Samplers for Particulates and Gases. Presented at the International Symposium on Indoor Air Pollution, Health and Energy Conservation, John F. Kennedy School of Government, Harvard University, Amherst, Massachusetts, October 13-16, 1981.

Medinsky, M. et al. A Similation Model Describing the Metabolism of Inhaled Selenium Compounds. Toxicology and Applied Pharmacology, 59:54-63, 1981.

A pharmacokinetic model for selenium metabolism is used to predict blood and organ selenium concentration in humans. The author concludes that selenium intake from air probably does not contribute significantly to the total human body burden, which appears to be derived mainly from dietary sources of selenium.

Mendez, W. M., Jr. and D. Hattis. Analysis of Available Evidence on Blood Lead-Air Lead Relationships Relevant to the Selection of a Permissable Exposure Limit for Lead in Air. Center for Policy Alternatives, Massachusetts Institute of Technology. CPA-78-13, June 1978. 146 pp.

Miller, C. Exposure Assessment Modeling: A State-of-the-Art Review. EPA-600/3-78-065, U.S. Environmental Protection Agency, Environmental Research Laboratory, Office of Research and Development, Athens, Georgia, July 1978. 56 pp.

The state-of-the-art review of exposure assessment modeling describes currently available models that simulate the environmental fate of substances, the exposure to such substances, and the effects of such exposure. The focus is first on exposure and then effects, where relatively little work has been done, and then on models of environmental fate, in particular on intermedia transfer processes. Single-medium air and water quality transport models are not assessed, but the possibility of approaching multi-media problems through a combination of single-medium approaches is explored. The report also describes several actual risk assessments that have been made using limited data and considers some secondary applications of the models. The investigation shows that the available models do not cover all of the areas necessary for an exposure assessment. More effort has been directed to modeling environmental fate than to modeling exposure and effects.

Mintz, S., R. Hosein, B. Batten, and F. Silverman. Design of a Personal Sampler of Three Respiratory Irritants. Presented at the International Symposium on Indoor Air Pollution, Health and Energy Conservation, John F. Kennedy School of Government, Harvard University, Amherst, Massachusetts, October 13-16, 1981. Abstract of Presentation.

The MITRE Corporation. Air Pollution Assessment of __________. EPA Contract No. 68-02-1495/077B. McLean, Virginia, 1976.

Benzene(Walker, P.)
Benzene acts primarily as a central nervous system depressant although it has also been shown to cause hematopoietic dysfunction and myelotoxicity in humans and animals. Production of benzene in the U.S. can utilize both coal and petroleum; however, the majority of the production is petroleum derived. Exposure of the human population to benzene can ensue not only from production facility emissions, but from usage of the compound, from oil spills, gasoline evaporation, and coke oven emissions. Control technology has been developed which, if utilized, can provide protection to industrial workers.

Carbon Tetrachloride(Johns, R.)
This report concerns the organochlorine solvent carbon tetrachloride, which is used primarily in the manufacture of fluorocarbons. The toxic effects of the solvent in both animals and man are discussed and the probability of those effects occurring at the ambient atmospheric concentrations to which the general population is exposed is assessed. Methods of atmospheric sampling of carbon tetrachloride and available control technology for the prevention of release to the environment are also discussed.

Cresols(Gordon, J.)
Cresol is a methyl substituted, hydroxylated aromatic hydrocarbon that occurs in three isomeric forms. Cresols are refined from coal tar or petroleum; they can also be synthesized. Cresols are toxic to both fauna and flora. Poisoning of humans has resulted from inhalation, ingestion, and absorption of cresol through skin and mucous membranes. Cresol affects the central nervous system, the kidneys, liver, lungs, heart, and stomach. Some fatalities and also carcinogenicity have been attributed to cresol. The principal sources of cresol emissions to the atmosphere are the production of cresols and the production of coke. Adsorption onto activated carbon is the best method for controlling the emission of cresol vapors into the atmosphere.

Ethylene Dibromide(Johns, R.)
Ethylene dibromide, a colorless, nonflammable hydrocarbon, is primarily employed as an additive in leaded gasoline to scavenge lead oxide residues from the combustion chambers of gasoline engines. Atmospheric measurement made near major suspected emission sources (service stations, refineries, production facilities) reveal ethylene dibromide concentrations several orders of magnitude below the threshold limit

value of 25 ppm established by the American Conference of Governmental Industrial Hygienists. Ethylene dibromide generally acts as a central nervous system depressant, although it is not as effective as many of the other halohydrocarbons.

Ethylene Dichloride(Johns, R.)
Ethylene dichloride, a chlorinated hydrocarbon, is primarily used as an intermediate during the production of vinyl chloride and other commercially valuable compounds. The characteristic water solubility and vapor pressure of ethylene dichloride indicate that this compound will tend to presist in the hydrosphere and lithosphere; while its slow activity with peroxide radicals and ozone indicates atmospheric persistence as well. Industrial exposure is limited by Occupational Safety and Health Administration regulations to 200 $\mu g/m^3$ (50 ppm). Ambient atmospheric measurements are not readily available. Inhalation of ethylene dichloride during acute exposure has been shown to produce central nervous system disorders as well as pathological effects in the liver, kidneys, and adrenals of humans, while chronic human exposure produces similar results.

Tetrachloroethylene(Fuller, B. B.)
Tetrachloroethylene, commonly known as perchloroethylene, is a synthetic halogenated hydrocarbon with excellent solvent properties. The primary physiological effect of tetrachloroethylene inhalation is central nervous system depression with loss of consciousness and death occurring at high concentrations. Liver damage has also been reported but only at or near lethal levels. The probability of environmental contamination is greatest at dry cleaning facilities where heated solvent vapors may be released to the atmosphere. Although adequate technology for the prevention of these losses is available, only 25% of the dry-cleaning establishments use such control. At metal degreasing facilities proper equipment design and adequate ventilation are sufficiently effective in maintaining safe vapor levels. The high volatility and low solubility of tetrachloroethylene are responsible for the entry of the solvent into the atmosphere. Tetrachloroethylene photodegrades in sunlight with a half life of 2 days, and is therefore not expected to accumulate in the environment. Concentrations in the ambient air over the highly industrialized Los Angeles Basin averaged 1.25 ppb but levels over rural areas and the open ocean averaged only 20 ppt.

Trichloroethylene(Fuller, B. B.)
Trichloroethylene is an organic solvent primarily used for the vapor degreasing of metals. Approximately 200,000 industrial workers are exposed to this solvent annually. Although the major physiological response in humans from acute or chronic exposure to trichloroethylene is central nervous system depression, damage to liver, kidney and heart have also been reported. Since the metabolic fate and toxic effects of trichloroethylene are similar in many mamalian species to those in man, the fact that this compound has recently been implicated as a potent liver carcinogen in mice may be of significance. Approximately 60 percent of the total world production of trichloroethylene is released to

the environment each year. However, due to its low water solubility, high vapor pressure, and high atmospheric photo degraduation rate, trichloroethylene is not expected to persist in the environment. Ambient concentrations in the atmosphere of industrialized areas are only about 16 ppt. Proper use of local exhaust systems in conjunction with vapor condensation apparatus and good general ventilation should be sufficient to maintain levels of trichloroethylene in the workshop environment well below the recommended 100 ppm and to insure a minimum release to the ambient atmosphere.

Toluene(Walker, P.)
Toluene, a homolog of benzene, is primarily a central nervous system depressant in both humans and animals. Only inconclusive evidence for hematopoietic or myelotoxic dysfunction was found. Toluene is produced in the U.S. from both coal and petroleum; however, the majority of this production is petroleum derived. Exposure of the human population to toluene can ensue not only from production facility emissions, but also from gasoline evaporation or automobile and coke oven emissions. The average concentration of toluene in urban air is reportedly 0.01 to 0.05 ppm. Control technology has been developed which, if utilized, can provide protection to industrial workers.

The MITRE Corporation. An Assessment of Cadmium in Drinking Water from a Multi-Media Perspective, 1979.

The study reviews the literature on environmental sources of cadmium exposure, absorbtion and pharmacokinetics and human dose-effect relationships. The relative importance of exposure to cadmium by various routes of exposure is assessed.

The MITRE Corporation. The Environmental Lead Problem: An Assessment of Lead in Drinking Water from a Multi-Media Standpoint. EPA-570/9-79-003, U.S. Environmental Protection Agency, Criteria and Standards Division, May 1979, 147 pp.

Human exposure to lead has been shown to be cumulative in nature. In order to assess the toxicological significance of environmental lead exposures, it is necessary to define the contributions to an individual's daily lead uptake from all possible exposure pathways. This paper defines and quantifies the major environmental sources of lead exposure, describes the absorption characteristics of lead compounds in man via each exposure route, determines the source contribution factors for daily lead uptake by each exposure pathway, and relates those contributions to an individual's blood-lead level.

Mockler, B. V., B. A. Wang, and M. J. Snow. Respirable Particulates Generated by Pressurized Consumer Products. I. Experimental Method and General Characteristics. American Industrial Hygiene Association Journal, 40(4):330-338, April 1979.

The simulated breathing zone approach to aerosol characterization for various commonly used pressurized household and personal care products has permitted collection of data under a variety of conditions. The principle exposure under actual use conditions is apparently to the aerosol that persists in the area after product discharge. The aerosols tested were collected during the 12 second product discharge burst and for 48 seconds afterward. Concentrations of particulates < 6 µm were 750 $mg/m^3$ in the breathing zone, determined under the worst reasonable conditions. Worst reasonable conditions exaggerate likely use concentrations by at least 2-6 times.

Moore, M. R. A Heavy Conference on Children and Lead. Ambio, 8(5):227-229, 1979.

Much remains to be understood regarding threshold Pb levels and neurophysiological effects. Soft water areas where Pb piping is used have brought about changes in blood-Pb concentrations. Lead smelters can cause nerve dysfunction and impairment of cognitive and peripheral motor function. Studies using electron probe microanalysis of electron microscopy sections of the brain showed clear accumulation of Pb within the mitochondria within the brain and, therefore, the dangers to brain energy metabolism constituted by such exposure. There is considerable variability in response to Pb exposure. The amount of permitted Pb in air is 1.5 $\mu g/m^3$ of air. The cost of controlling exposure is high, but the cost of human damage is equally high. The measurement of protoporphyrin levels may be required to control both Pb poisoning and Fe deficiency anemia. Tooth Pb levels reflect an overall analysis of Pb poisoning, while blood Pb levels indicate recent exposure.

Morrow, P.E. Pulmonary Retention of Lead in Man: An Experimental Study. Environmental Research, 21:373-384, 1980.

The half life for retention of small particle (MMAD 0.25µm) of soluble lead aerosol in the lung was measured in 17 subjects by lead-203 counting. The observed efficacy of deposition was 23 to 26 percent and the half-times for retention in the lung were between 13 and 14 hours for the two experimental groups.

Moschandreas, D. J. and Stark, J. W. (Geomet, Inc.) The GEOMET Indoor-Outdoor Air Pollution Model. EPA-600/7-78-106, United States Environmental Protection Agency, Environmental Monitoring and Support Laboratory, Research Triangle Park, North Carolina, June 1978.

This report documents the formulation of the GEOMET Indoor-Outdoor Air Pollution (GIOAP) model. The model estimates indoor air pollutant concentrations as a function of outdoor pollutant levels, indoor pollutant generation source rates, pollutant chemical decay rates, and air exchange rates. Topics discussed include basic principles, model formulation, parameter estimation, model statistical validation, and model sensitivity to perturbations of the input parameters.

The numerical estimates obtained from the GIOAP simulations have been validated with observed hourly pollutant concentrations obtained from an 18-month residential air quality sampling program. Statistically, the model values of carbon monoxide are within 10% of the observed values in the concentration range interval which includes 85% of all hourly measurements. Similarly, for nitric oxide the predicted values are within 15% of the observed; for nitrogen dioxide the difference between the predicted value and the ideal condition of exactly estimating the corresponding observed value is 16% for 85% of the observed hourly concentrations. Also, the GIOAP model predicts within 25% for nonmethane hydrocarbons and within 8% for carbon dioxide. Thus statistically the GIOAP model estimations are within 25% of the ideal condition (estimated and observed values coincide) for 85% of the observed values for CO, NO, $NO_2$, NMHC, and $CO_2$. The model has not been validated against sulfur dioxide due to the very low values measured both indoors and outdoors. The predetermined validation criteria were not satisfied by the ozone model estimations. The calculated values, however, were judged adequate because for about 85% of the observed values the predicted concentrations were within 2 ppb.

Moschandreas, D. J. and Stark, J. W. (Geomet, Inc.) The Residential Environment and Energy Conservation: Predicting Indoor Air Quality. Presented at the 71st Annual Meeting of the Air Pollution Control Association, Houston, Texas, June 25-30, 1978. 14 pp.

The GEOMET Indoor-Outdoor Air Pollution (GIOAP) model predicts indoor pollutant concentrations as a function of outdoor levels, indoor pollutant sources and sinks, and the air exchange rate. The GIOAP model, based on a mass balance principle, has been validated on the data base generated by the field experiments of an 18 month monitoring program of residences. It predicts indoor pollution concentrations within 20 percent of the observed hourly indoor values for carbon monoxide (CO), nitrogen oxide (NO), nitrogen dioxide ($NO_2$), carbon dioxide, and total hydrocarbons-methane. A series of numerical simulations using the GIOAP model show that under certain conditions involving gas appliances, indoor CO, NO and $NO_2$ concentrations increase substantially. Under these same conditions carbon monoxide concentrations reach levels that violate the hourly ambient national standard. This is of particular current concern because this violation may be attributed to energy conservation measures and the resulting decrease in the air exchange rates. The dynamics of energy conservation and indoor residential air quality are investigated and a series of optimization procedures are discussed by predicting the indoor pollutant concentrations as a function of varying air exchange rates, indoor emission rates, and filtering devices.

Moschandreas, D. J., J. W. Stark, et al. Indoor Air Pollution in the Residential Environment. EPA-600/7-78-299a, U.S. Environmental Protection Agency, Office of Research and Development, Environmental Monitoring and Support Laboratory, Research Triangle Park, North Carolina, December 1978. 182 pp.

A 24-month study was undertaken to characterize the indoor residential air quality. Seventeen residential dwellings were monitored, each for a 14-day period. Air samples were collected from four locations: one outdoor site adjacent to the building; and three indoor sites, the kitchen, bedroom, and living room. "Continuous" sampling was carried out for CO, $SO_2$, NO, $NO_2$, $CO_2$, $O_3$, $CH_4$, and THC, TSP, RSP, $SO_4$, $NO_3$, Pb, ammonia, and aldehydes were monitored intermittently. Aerosol samples were collected for elemental analysis by the Proton Induced X-ray Emission (PIXE) technique. In addition, data on energy parameters, infiltration rates, and family activities were obtained by observations, field experiments, and daily questionnaires, respectively. Each residence was monitored with minimal interference in the daily activities of the occupants; thus, the residential pollutant concentrations were determined under real-life conditions.

The air quality of the residential environment was determined to be markedly different from the outdoor ambient air quality. Three classes of air pollutants have been identified with respect to indoor-outdoor pollutant relationships: 1) concentrations of CO, NO, $CO_2$, NMHC, and aldehydes in the residential environment are often higher than outdoors; 2) TSP and RSP are sometimes lower and sometimes higher indoors than outdoors; and 3) indoor concentrations of $SO_2$, $O_3$, $SO_4$, and $NO_3$ are almost always lower than the corresponding outdoor pollutant concentrations. Indoor concentrations of TSP, $O_3$, NMHC, and CO have been observed to equal or exceed the National Ambient Air Quality Standards for these pollutants.

Moschandreas, D. J., J. W. Winchester, J. W. Nelson, and R. M. Burton. (GEOMET, Inc.) Fine Particle Residential Indoor Air Pollution. Atmospheric Environment, 13(10):1413-1418, 1979.

Indoor-generated elemental constituents of fine aerosol particulate matter may be distinguished from constituents infiltrating from the outdoors by comparing time variation in concentrations hourly. Measurements within 3 residential buildings indicate that S and Pb-Br aerosols are predominantly of outdoor origin, although a secondary indoor Pb source was detected. Potassium is, to a significant degree, of indoor origin. Indoor-outdoor exchange times inferred for fine particle constituents agree with those estimated for gas exchange and with model calculations, suggesting conservative fine aerosol transport. However, coarse particle constituents, e.g., Fe, exhibit time variability complicated by nonconservative behavior, such as removal by filtration and settling.

Moschandreas, D. J. Indoor Air Pollution in the Residential Environment--Volume II: Field Monitoring Protocol, Indoor Episodic Pollutant Release Experiments and Numerical Analyses. EPA-600/7-78-229b, U.S. Environmental Protection Agency, Office of Research and Development, Environmental Monitoring and Support Laboratory, Research Triangle Park, North Carolina, December 1978. 231 pp.

Murano, D. B., E. P. Savage, and R. M. Buchan. Altitude Affects Carbon Monoxide Formation in Pickup Campers. Journal of Environmental Health, 40(4):198-202, January-February 1978.

To evaluate the effect of increased altitude on the amount of carbon monoxide (CO) produced by gas-fired appliances, 20 pickup campers were tested for CO concentration above 8,500 ft., and retested below 5,500 ft. The mean CO concentration in the campers at low altitude was 38 ppm, compared to 57 ppm at high altitude; the increase can be attributed to inefficient combustion of gas appliances due to reduced $O_2$ availability at the higher altitude. The magnitude of the CO problem was related to camper age; the mean CO concentrations were 12 ppm (low altitude) and 18 ppm (high altitude) for campers less than 5 yr old, 49 and 75 ppm for those 5-10 yr. old, and 65 and 102 ppm for those over 10 yr. old. The problem of greater CO production at high altitude is compounded by altitude hypoxia, which makes the occupants less tolerant of CO. Of the 30 campers tested, 13 exceeded the EPA 1-hr. exposure standard of 35 ppm.

Nagda, N. L. (GEOMET, Inc.) Environmental Carcinogens and Human Cancer--Estimation of Exposure to Carcinogens in the Ambient Air. EPA-600/1-79-002. U.S. Environmental Protection Agency, Office of Research and Development, Health Effects Research Laboratory, Research Triangle Park, North Carolina, 1979. 155 pp.

In this study, a methodology for ambient exposure analysis of carcinogens was developed based on a pilot study of the Detroit Metropolitan area. The specific aim of the analysis was to identify high and low exposure areas within the study area. Four known or suspected carcinogens and groups of carcinogens: BaP, trichloroethylene, nickel and its compounds, and cadmium and its compounds were studied. The analysis of ambient exposure to BaP consisted of the use of the Air Quality Display Model (AQDM) to simulate levels of BaP which might have existed during 1956 to 1960. The analysis for BaP involved a multistep procedure. In order to examine recent conditions, the years 1975-1976 were simulated and compared against known concentrations in the area. Next, BaP emissions for the period 1956-1960 were estimated by analyzing past trends for significant sources. This emissions data base, along with meteorological data for the same period, was used as an input to ADQM to predict historical exposure to BaP. The analysis for the other three carcinogens was less detailed than that for BaP. It consisted of estimating emissions and calculating an emission density for each of the three carcinogens. For nickel and cadmium, it also included a comparison of spatial variation in emissions with measured air quality patterns in the Detroit area.

Nagda, N. L. and L. R. Babcock, Ranking Air Pollution Sources by Population Exposure: Health Effects Assessment. Presented at the 71st Annual Meeting of the Air Pollution Control Association, Houston, Texas, June 25-30, 1978. 15 pp.

The problem of incorporating multiple-pollutant health-effects information into source-receptor policy-analyses is addressed. A literature analysis was undertaken to assess the feasibility of deriving dose-response relationships from results of experimental and epidemiological studies which utilized a variety of pulmonary function tests. Twenty-three studies were analyzed, categorized, and compared in various ways. Many of the studies appeared deficient in that variables which significantly affected pulmonary function tests were neither quantified nor held constant. A reliability index was developed and used to rank existing studies. Despite some progress and presence of voluminous data, gaps and uncertainties are so extensive that existing knowledge must be supplemented with additional well-designed experimental and epidemiological studies before meaningful dose-response curves can be developed and utilized. Tolerance-factor methods involving extrapolation of existing ambient air quality standards (e.g. pindex") presently provide the most workable means of integrating multiple-pollutant health effects into simulations and other policy-analysis studies..

National Academy of Sciences, Committee on Lead in the Human Environment, Lead in the Human Environment, Washington, D.C., 1980.

Nitta, H. and K. Maeda. Personal Exposure Monitoring to Nitrogen Dioxide. Environment International, 8:243-248, 1982.

Ott, W. An Urban Survey Technique for Measuring Spacial Variation of Carbon Monoxide Concentrations in Cities. Ph.D. Dissertation, Stanford University, Department of Civil Engineering, 1971.

Ott, W.R. and Mage, D.T. Interpreting Urban Carbon Monoxide Concentrations by Means of a Computerized Blood COhb Model. Journal of the Air Pollution Control Association, 28:911-916, 1978.

Ott, W. R. Exposure Estimates Based on Computer Generated Activity Patterns. Prepared for presentation at the 74th Annual Meeting of the Air Pollution Control Association, Philadelphia, Pennsylvania, June 21-26, 1981. 16 pp.

A computer program called Simulation of Human Air Pollution Exposures (SHAPE) was written which uses Monte Carlo simulation to calculate carbon monoxide (CO) exposures of the population. During a 24-hour period, the program assigns each person to specific "microenvironments" (for example, home, automobile, office, garage) based on probability distributions derived from activity pattern studies. The program then combines activity profiles with data on CO concentrations in specific microenvironments and urban background concentrations in order to calculate integrated exposures of a large number of persons over a 24-hour period. Instead of a Markov chain approach, both the time spent engaged in a specific activity and the transitions from one activity to another are treated as random variables that are sampled from distributions specified by the user. The time resolution of SHAPE is on a minute-by-minute basis, and activities (and concentrations) are coded into an activity vector consisting of 1440 minutes per 24-hour period.

The "inverse transformation method" for generating random variables is used, so any frequency distribution can be employed as an input to the program. The SHAPE program was run for 400 persons using activity pattern data collected by the U.S. Census for the Department of Transportation and concentration data from empirical field studies and an urban air monitoring station. The resulting frequency distributions of maximum hourly CO exposures and maximum moving average 8-hour exposures for 400 persons illustrate the methodology.

Ott, W. R. Models of Human Exposure to Air Pollution. Technical Report No. 32, July 1980. Prepared under the auspices of Siam Institute for Mathematics and Society.

This paper lays out a mathematical framework for defining and characterizing exposure to air pollutants. The meaning and utility of average exposure over different averaging periods is discussed, along with problems in defining pollutant exposure over time for individuals and populations.

Ott, W. and P. Flachsbart. Measurement of Carbon Monoxide Concentrations in Indoor and Outdoor Locations Using Personal Exposure Monitors. Environment International, 8:295-304, 1982.

Palmes, E. D., C. Tomcyzk, and A. W. March. Relationship of Indoor $NO_2$ Concentrations to Use of Unvented Gas Appliances. Air Pollution Control Association Journal, 29(4):392-402, April 1979.

Passive air samplers were used to monitor homes in Gainesville and in surrounding small towns and rural areas of Florida. Participants taped the samplers on the wall at eye level in the kitchen 2-5 ft. from the space heater, and in an unheated living space. Exposures lasted 1 wk. In the group where there were no unvented space heaters, the $NO_2$ concentrations were quite low, averaging only 3 ppb. Where the only unvented gas appliance was the kitchen stove, the kitchen concentration was 55 ppb, and 2 other rooms without heaters averaged 28 and 19 ppb. In another group, where a heater was the only unvented appliance, the $NO_2$ concentration in the kitchen and in the room containing the heater was 11 ppb, with the unheated living space equal to 1/2 this value. Where there was a gas stove in the kitchen and a heater in another room, the concentration was 150 ppb in both rooms, and 1/2 that in an unheated room. Where there was a heater in the kitchen and in another room, the concentration in both rooms was far higher than any other group, showing that the concentration of 2 heaters was additive. In spite of the small sampling, the differences in $NO_2$ concentrations are so striking and consistent that the results are meaningful and add another warning note concerning the potential hazard of unvented space heaters.

Pellizzari, E.D., et al. Total Exposure Assessment Methodology (TEAM) Study: Phase II, Part II: Protocols for Environmental and Human Sampling and Analysis. Office of Monitoring, System and Quality Assurance, U.S. Environmental Protection Agency.

This document describes methods to be used to measure the concentration of a wide variety of organic compounds and heavy metals in body fluids to be used in Phase II of the TEAM study.

Peterson, J.E. Modeling the Uptake, Metabolism and Excretion of Dichloromethane by Man. American Industrial Hygiene Association Journal, 39(1):41-47, January 1978.

The effects of human exposure to concentrations of dichloromethane, a paint and varnish remover, were studied. Experimental methods and materials used are described. Data were obtained from the exposure of 11 men to dichloromethane concentrations of 50, 100, 250, and 500 ppm, 1,3, or 3.5 hr/day for as long as five successive days, and from the exposure of nine women to 250 ppm on a similar schedule. Breath concentrations of the solvent were related mathematically to exposure parameters, as were blood concentrations of the metabolite, carbon dioxide. The resulting empirical equations can be used to predict the consequences of many industrial and nonindustrial exposure situations. No acceleration of dichloromethane metabolism was found, indicating that effective enzyme induction did not occur during five weeks of exposure.

Porstendoerfer, J. A. Wicke, and A. Schraub. The Influence of Exhalation, Ventilation, and Deposition Upon the Concentration of Radon (222Rn) and Thoron (220Rn) and their Decay Products in Room Air. Health Physics, 34(5):465-473, May 1978.

On the basis of a simple room model, the degree of equilibrium between 222 Rn and 220 Rn and their decay products was calculated considering the measured natural radioactive indoor and outdoor aerosol size distribution, the known attachment rate of the uncombined decay products, the experimentally evaluated recoil factor, and an estimated deposition rate on a wall surface. The indoor concentration is always greater than outdoors by a factor of >50. In contrast, the Rn concentration does not depend on ventilation, but is determined by exhalation from the building material's surface. The ratio of concentrations between Rn and the free and attached decay products indoors is influenced by ventilation, exhalation, and wall-deposition of the free radioactive atoms. Although the relative concentrations of Rn daughters are small indoors, their absolute values are high. In further investigations, the influence of the discussed parameters on the indoor concentration of 222 Rn, 220 Rn, and their daughters should be studied experimentally and the resulting lung exposure estimated.

Quant, F. R., P. A. Nelson and G. J. Sem. Experimental Measurements of Aerosol Concentrations in Offices. Environment International, 8:223-227, 1982.

Rafferty, P. J. Drinking Water Quality and Cancer Mortality Patterns: Progress Report #1, EPA Office of Drinking Water Standards.

Repace, J. and A. H. Lowrey. Indoor Air Pollution, Tobacco Smoke, and Public Health. Science, 208(4443)464-472, May 1980.

Research that assessed the health hazards to nonsmokers posed by exposure to tobacco smoke in buildings is described. A model that incorporates both physical and sociological parameters in determining the levels of sociological parameters in determining the levels of respirable suspended particulates in tobacco is developed. Results indicate that nonsmokers are exposed to significant air pollution burdens from indoor smoking. The public health implications of this exposure is assessed.

Ricci, L. J. New Health Monitors: Small But Accurate. Chemical Engineering, 86(28):28-29, December 31, 1979.

A program for the development of portable personal exposure monitors is being funded by the EPA. A miniature GC which incorporates a microcomputer is among the new developments and has been designed to analyze 90 chemicals and to weigh only 1.3 lb. Commercial units will, however, probably cost more than the originally estimated $2,000, so first applications are unlikely to be for routine monitoring. Another instrument reaching the field testing stage is a portable X-ray fluorescence unit for onsite nondestructive analysis of approximately 25 elements. For most conditions the unit is expected to be accurate to within plus or minus 105, but the capital cost may be as much as $10,000-15,000. The fibrous Aerosol Monitor, developed for the asbestos industry weighs 27 lb. Field results correlate with standard laboratory methods for asbestos; however, interference from nonfibrous dust restricts the device to applications in which such concentrations are less than several milligrams per cubic meter. Personal devices for monitoring exposure to UV radiation have not been very successful. A monitor based on a chemical actinometer system must be further evaluated before the devices can approach commercial status.

Richter, E. D., Y. Yaffee, and N. Gruener. Air and Blood Lead Levels in a Battery Factory. Environmental Research, 20(1):87-98, October 1979.

Blood Pb (PbB) and air Pb (PbA) exposure levels were studied in 62 workers in a battery plant. Thirty-five battery workers had PbB of $> 60\ \mu g/100$ ml (OSHA Proposed Biologic Limit Value, (BLV), 1975). Fifty-eight workers had PbBs of $> 40\ \mu g/100$ ml (OSHA Proposed BLV, 1978). Average PbA for 33 battery workers was $< 100\ \mu g/m^3$, the newly recommended time-weighted average. Average PbAs of approximately 50 $\mu g/m^3$, (6 workers), 85 $\mu g/m^3$ (17 workers), and 190 $\mu g/m^3$ (10 workers) were associated with virtually identical mean PbB values (44.0, 59.5, and 58.4 µg/100ml). For 10 of 29 battery workers, the PbA value (< 100 µg/m$^3$) was associated with individual PbB values (> 60 µg/100 ml). At exposure levels of 329 µg/m$^3$ (15 workers), 885 µg/m$^3$ (3 workers), and 1,187 µg/m$^3$ (4 workers), the associated mean PbB was 75.2, 76.3, and 90.7 µg/100 ml, respectively. Complaints of fatigue, headache, abdominal pain, and loss of appetite were more frequent among 35 workers with PbB of > 60 µg/100 ml compared with 26 workers with PbB of < 60 µg/100 ml (relative risk 1.5, 1.4, 1.9, and 1.9, respectively). The relationship between group PbA and individual PbB was expressed mathematically. Battery factory data do not support the assumption that if Pb A were kept at 100 µg/m$^3$, the blood Pb levels would be < 60 µg/100 ml.

Roberts, J. R. and W. K. Marshall. Retentive Capacity: An Index of Chemical Persistence Expressed in Terms of Chemical-Specific and Ecosystem-Specific Parameters. Ecotoxicology and Environmental Safety, 4(2):158-171, June 1980.

Retentive Capacity (RC) is defined as the mean residence time of a particular chemical in an ecosystem and is determined by the characteristics of the ecosystem, e.g., pH, SS, and biomass, and by the characteristics of the chemical, e.g. hydrolysis rate, vapor pressure, and solubility. In defining RC, first order kinetics are assumed. The information obtained in the development of a persistence screen can be used to assess monitoring methods for field residues and for conducting preliminary assessments of exposure patterns. To establish a framework with which the relevance of toxicity testing and monitoring studies can be assessed, it is necessary to examine the validity and limits of the equilibrium assumption, to determine the effective depth of the sediment pool, and to establish the intrinsic factors characterizing the sorptive properties of the pools.

Roels, H. A., J. P. Buchet, and R. Lauwerys. Lead and Cadmium Absorption Among Children Near a Nonferrous Metal Plant: A Follow-Up Study of a Test Case. Environmental Research, 15(2):290-308, April 1978.

Eighteen months after major improvements were introduced in a Pb smelter to reduce its Pb emission, a second survey was carried out in the same schools (with comparable groups of children) as an initial survey. Continuous air monitoring revealed a considerable decrease in airborne Pb for the Pb smelter area. The median values dropped from 3.2 to 1.2 µg Pb/m$^3$ at < 1 km and from 1.6 to 0.5-0.8 µg Pb/m$^3$ at 1.5 km from the plant; in the rural area it remained unchanged (3.0 µg Pb/m$^3$). The Pb and Cd exposure of the children were assessed by measuring blood Pb (PbB) and urine Pb (PbU), erythrocyte d-ALAD activity, free erythrocyte porphyrine (FEP) concentration, d-ALAD excretion in urine (ALAU), and Cd in blood (CdB) and urine (CdU). Subgroups A and B (< 1 km distant and 2.4 km distant, respectively) exhibited higher PbB and CdU levels, and lower ALAD activity than the rural group. However, PbU, CdB, FEP, and ALAD were significantly increased only in subgroup A. Compared with the results of the first survey, a significant tendency to the normalization of PbB was found at 2.5 km, but at < 1 km the biological parameters (particularly PbB, ALAD, FEP) did not improve.

Rosenblatt, David H. and David R. Cogley. An Environmental Fate Model Leading to Preliminary Pollutant Limit Values for Human Health Effects. Environmental Risk Analysis for Chemicals, Richard Conway, Ed. VanNostrand Reinhold Company, New York, 1980.

Roth, P., S. D. Reynolds, P. J. W. Roberts, J. H. Seinfeld. (Systems Applications, Inc.) Development of a Simulation Model for Estimating Ground Level Concentrations of Photochemical Pollutants. Report 71SAI-21, prepared for the Air Pollution Control Office of the Environmental Protection Agency, Durham, North Carolina, July 1971.

This report describes the progress that has been achieved to date in the development and validation of a simulation model for estimating ground level concentrations of photochemical pollutants. The bulk of the effort reported here is developmental, involving the compilation of a comprehensive source emissions inventory, the development and validation of the method of fractional steps for use in the solution of the governing equations, and the preparation of maps displaying spatial and temporal variations in wind speed and direction and in the height of the inversion base. The details of these various efforts are described in a series of appendices to this report. Although a validated kinetic mechanism has been developed and incorporated in the simulation model, validation efforts have thus far been restricted to carbon monoxide. Provisional validation results for the Los Angeles Basin are presented.

Rupp, E. M. Age Dependent Values of Dietary Intake for Assessing Human Exposures to Environmental Pollutants. Health Physics, 39:151-163, August 1980.

Detailed information was compiled concerning human food consumption patterns necessary for relating pollutant body burdens to environmental contamination levels. It was to be used in lieu of site-specific data for calculating health impacts arising from the ingestion of food containing various levels of such pollutants as radionuclides, toxic heavy metals, insecticides, and many others. Impact assessments for which food ingestion data are necessary may include pollutants from such diverse sources as mine tailings; construction and operation of power plants (coal, nuclear, gas geothermal, etc.); recovery of uranium from phosphoric acid; decontamination and decommissioning of nuclear facilities; food additives; industrial and agricultural activities; and methods of disposal of various kinds of waste materials.

This paper examines available data on human food consumption rates and estimates dietary intake rates by individuals of various ages. Ingestion parameters include intake rates of water and other beverages, milk, vegetables, fruits, meat, poultry, grain and fish. Information was analyzed from documented surveys of individuals within households and from interviews and personal communications. Although average intake rates are often the only data reported in the literature, ranges as well as averages are included in this paper whenever possible.

Various results are tabulated, including per capita food consumption in 1955 and 1965, changes in per capita consumption, and changes by urbanization. Details of milk consumption are presented by selected sex-age groups. Solid food intake is given by age group. Market basket quantities of foods consumed daily are also tabulated.

Rupp, E. M., *et al*. Composite Hazard Index for Assessing Limiting Exposures to Environmental Pollutants: Application through a Case Study. *Environmental Science and Technology*, *12*, 802-807, July, 1978.

The procedure described by Walsh (1978) is used to derive limiting concentrations of Cadmium in air near a smelter.

Rydell, C. P., D. Collins, A. Dedrick, H. Kaminski, and G. Schwarz. Air Pollution and Urban Population Distribution. Hunter College Research Center, CUNY, under U.S. Public Health Service Grant No. AP 00555, July 1968. 72 pp.

When air pollutant generation is assumed to be proportional to population density, a nearly uniform urban population distribution indicates minimal pollution damage. Subjects discussed are: Population distribution which minimizes damages from area sources with emissions proportional to population density; population distribution which minimizes damages from area sources with emissions a linear transformation of population density; population distribution that equalizes per capita air pollution damages; and the effect of air pollution costs on optimal urban format. Appendices are included on air pollution generation from area source, air pollution diffusion models, air pollution damage evaluation, urban population distribution explained by density cost functions and population distribution calculations that minimize a quadratic cost function.

Santodonato, J., D. Basu, and P. Howard. Health Effects Associated with Diesel Exhaust Emissions; Literature Review and Evaluation. EPA-600/3-78-065, U.S. Environmental Protection Agency, Office of Research and Development, Health Effects Research Laboratory, Research Triangle Park, North Carolina, November 1978. 154 pp.

Schwarz, G. Local, Regional, and Global Collective Dose Commitments from Gaseous Airborne Effluents. *Health Physics*, 37(6):827-828, December 1979. Abstract Only.

Airborne radionuclides are dispersed in the atmosphere and are deposited on the soil surface or vegetation according to their physicochemical behavior. With respect to the evaluation of the radiological implication of single nuclides, a few nuclides represent not only a hazard by first pass exposure pathways but also an additional hazard due to their global distribution. Previous consideration of this fact led to the development of the collective dose commitment concept. Collective dose commitments are discussed for various nuclides with importance to the environment. Emphasis is placed on the dose commitments received by distinct populations from gaseous effluents released within the Federal Republic of Germany. The total dose commitment may be estimated by summing the dose commitments for these populations. For releases of $^{14}C$ and $^{85}Kr$ the global contribution to the total dose commitment is 99 percent and 86 percent, respectively. For $^{3}H$, however, the global contribution is relatively small, around 7%. For most other nuclides, only local and regional collective dose commitments are of importance.

Sega, K. and M. Fugas. Personal Exposure Versus Monitoring Station Data for Respirable Particles. Environment International, 8:259-263, 1982.

Spengler, J.D., D.W. Dockery, W.A. Turner, J.M. Wolfson, and B.G. Ferris Jr. Indoor-Outdoor Air Pollution Relationships: Results of the Indoor-Outdoor Monitoring Program of the Harvard Six City Study. 72nd Annual Meeting of the Air Pollution Control Association, Cincinnati, Ohio, June 24-29, 1979.

The results of extensive indoor and outdoor monitoring for sulfur dioxide, nitrogen dioxide, respirable size particles, and the sulfate fraction of these particles are reported. Indoor mass concentrations of respirable particles are higher than the outdoor levels. The major source of indoor particulate matter is cigarette smoke. While the outdoor concentrations are significantly different among the six cities of this study, there is not a significant difference among sites within cities except for one heavily industrialized city. The analysis of variance on indoor concentrations indicates a significant difference among cities and among sites within cities.

The indoor respirable sulfate concentrations are lower than the outdoor concentrations with an overall ratio of .7. Sulfur dioxide concentrations are much lower inside homes than outside. In higher $SO_2$ areas overall reductions of 50% are typical. In the absence of air conditioning the indoor/outdoor $SO_2$ ratio approaches unity in the summer months when windows and doors are open. For nitrogen dioxide the levels can be much higher indoors than outdoors. Gas appliances and lack of ventilation lead to the highest $NO_2$ levels.

Spengler, J.D., D.W. Dockery, W.A. Turner, J.W. Wolfson, and B.G. Ferris. Long Term Measurements of Respirable Sulfates and Particles Inside and Outside Homes. Atmospheric Environment, Vol. 15, pp. 23-30, 1981.

The results of extensive indoor and outdoor monitoring for respirable size particles and the sulfate fraction of these particles are reported. These air pollution measurements were obtained in conjunction with an epidemiologic study in six U.S. cities: Portage, Wisconsin; Topeka, Kansas; Kingston-Harriman, Tennessee; Watertown, Massachusetts; St. Louis, Missouri; and Steubenville, Ohio. The major source of indoor particulate matter is cigarette smoke, which contributes approximately 20 $\mu g/m^3$ to the indoor concentrations for each smoker. Even in homes without smokers, indoor particle concentrations equal or exceed outdoor levels. The indoor respirable sulfate concentrations are consistently lower than outdoor concentrations.

Spengler, J. D., B. G. Ferris, Jr. and D. W. Dockery. Sulfur Dioxide and Nitrogen Levels Inside and Outside Homes and the Implications on Health Effects Research. Environmental Science and Technology, 13(10):1276-1280, October 1979.

The results of 1 year's indoor and outdoor monitoring for $SO_2$ and $NO_2$ in 6 communities with widely varying outdoor levels are presented. The representativeness of the monitoring in defining exposure is discussed for each city. In four of the communities, outdoor $SO_2$ levels are less than 50% of the annual National Ambient Air Quality Standards, while violations are found in the other two. Annual average indoor concentrations are 20-70% of the outdoor levels. Indoor $NO_2$ levels can exceed outdoor levels by a factor of 2, depending on the type of cooking appliance used but do not exceed the standard. The impact of various heating and cooking systems on the indoor concentrations of these gases, as well as the type of appliances used in the house, is evaluated.

Spengler, J. D., R. D. Treitman, T. D. Tosteson, and D. T. Mage. Personal Exposures to Respirable Particulates: A Tale of Two Cities - Kingston and Harriman, Tennessee. Presented at the International Symposium on Indoor Air Pollution, Health and Energy Conservation, John F. Kennedy School of Government, Harvard University, Amherst, Massachusetts, October 13-16, 1981. Abstract of Presentation.

Spengler, J.D., and T. Tosteson. Personal Exposures to Respirable Particles. Presented at Environmetrics 81, Conference of the Society for Industrial and Applied Mathematics, Alexandria, VA, April 1981.

Spivey, G. H. and E. Sloss (UCLA School of Public Health). Cancer and Chlorinated Drinking Water, Part II. EPA Contract No. CA-6-99-3349-J, U.S. Environmental Protection Agency, Office of Research and Development, Health Effects Research Laboratory, Cincinnati, Ohio, May 1977. 70 pp.

Spivey, G. H. and M. R. Fox. Nutritional Influences on Metal Toxicity: Cadmium as a Model Toxic Element. Environmental Health Perspectives, 29:95-104, April 1979.

Adequate dietary nutrition is recognized as a significant factor in modifying the response of humans and animals to toxic element exposure. Nutrient deficiencies can exacerbate the effects of cadmium, and supplements of essential nutrients have been shown to ameliorate cadmium toxicity. The effects of exposure to a toxic element, such as cadmium, may vary depending on interactions with other elements that are present in the diet in different concentrations. Cases of cadmium toxicity in humans are reviewed. Discussed are studies on: the effects of dietary nutrients in increasing risks from cadmium; effects of dietary nutrients in decreasing risks from cadmium; effects of age on cadmium uptake; enteropathy caused by dietary cadmium; and nutritional intervention to protect against excess metal intake in humans.

Stark, A.D., et al. Family Operation Cofactors in the Epidemiology of Childhood Lead Poisoning. Archives of Environmental Health, 33:222-226, 1978.

Sterling, T.D. and D. Kobayashi. Use of Gas Ranges for Cooking and Heating in Urban Dwellings. Air Pollution Control Association Journal, 31(2):162-165, February 1981.

While several investigations have isolated kitchen gas ranges as a major source of indoor air pollution, such studies have only considered the normal operation of gas ranges for cooking. Two studies to assess the extent to which gas ranges are being used to heat urban dwellings are discussed. On the basis of gas consumption data and temperature measurements obtained in New York City during 1977, and actual observations of supplemental heating practices with gas ranges in 120 homes in the South Bronx, New York City, it is estimated that some 50-55% of New York City's gas ranges are used for both cooking and heating. Increased human exposure to carbon monoxide and nitrogen dioxide as a result of these practices is discussed.

Stopford, W., S. D. Bundy, L. J. Goldwater, et al. Microenvironmental Exposure to Mercury Vapor. American Industrial Hygiene Association Journal, 39(5):378-384, May 1978.

Ten individuals with known exposure to Hg vapor and five individuals with no known exposure to Hg vapor were monitored over a 5 day work-week. Environmental and microenvironmental Hg vapor samples were collected by passing air through tubes filled with Hopcalite. Desorbed Hg was analyzed with flameless atomic absorption spectrometry. Environmental Hg levels ranged from 8 to 88 $\mu g/m^3$ for the exposed population and concurrent microenvironmental levels ranged from 16 to 687 $\mu g/m^3$. Urine and blood Hg values were not correlated with environmental Hg levels. Total urine Hg values corrected for specific gravity were highly significantly correlated ($p < .001$) with microenvironmental Hg levels; blood Hg values also gave a good estimate of average microenvironmental exposure. The total urine Hg concentration of a single collection at the end of the work-week was as useful as the mean urine Hg concentration of samples collected over the entire week in estimating the mean Hg vapor exposure for the week. Elevated levels of Hg vapor above an average moderate exposure level were not reflected in unusual amounts of ionic or elemental Hg excreted in urine.

Todd, W. J., A. J. G. George, Jr. and N. A. Bryant. Satellite-Aided Evaluation of Population Exposure to Air Pollution. Environmental Science and Technology, 13(8):970-974, August 1979.

The ability to determine population exposure to air pollution through computer processing utilizing Landsat satellite-derived land use information is demonstrated. Three variables-a 1985 estimate of TSP for 2-Km$^2$ grid cells, Landsat-derived residential land cover data for 0.45-hectare cells, and population totals for census tracts-were spatially registered and cross-tabulated to produce tabular and map products illustrating relative air quality exposure for residential population by 2-km$^2$ cells. It would cost $20,000 to replicate the analysis for an area similar in size to the 4,000-km$^2$ Portland, Oregon, area. Once completed, the spatially fine, computer-compatible air quality and population data are amenable to the timely and efficient generation of population-at-risk tabular and map information on a continuous or periodic basis.

Turner, W.A., J.E. Spengler, D.W. Dockery, and S.D. Colome. Design and Performmance of a Reliable Personal Monitoring System for Respirable Particulates. Journal of the Air Pollution Control Association, Volume 29, No. 7, July, 1979, pp. 747-748.

Tuthill R. W. and G. Moore. Chlorination of Public Drinking Water and Subsequent Cancer Mortality: An Ecological Time-Lag Study. EPA Contract No. EPA-68-03-1200, U.S. Environmental Protection Agency, Health Effects Laboratory, Cincinnati, Ohio, May 1978. 35 pp.

U.S. Environmental Protection Agency. Analysis of Population Exposure to Air Pollution in New York-New Jersey-Connecticut Tri-State Region. EPA-450/3-76-027, U.S. Environmental Protection Agency, Office of Air and Waste Managment, Office of Air Quality Planning and Standards, Research Triangle Park, North Carolina, March 1976. 168 pp.

A population exposure methodology has been developed and applied to total suspended particulate (TSP) in the NY-NJ-Conn Tri-State Region. Ambient TSP data produced in 72 monitoring stations, 1971 to 1973, are used for the analysis of population exposure to TSP. Census data are aggregated into 215 points to form a demographic network. The monitored air quality data are spatially interpolated to each demographic network point to calculate a local population exposure.

Annual and quarterly geometric mean concentrations are used to estimate long-term population exposure to TSP. Long-term exposure is characterized by a population dosage spectrum that indicates a population distribution of exposures at various mean concentrations. Population average air quality is computed to indicate representative air quality levels. A health risk index indicates a percentage of the population exposed to air pollution above the annual standard.

U.S. Environmental Protection Agency. Assessment of Human Exposures to Atmospheric Benzene. EPA-450/3-78-031, U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards, Research Triangle Park, North Carolina, June 1978.

This report is one of three reports which were prepared by EPA to determine what regulatory action should be taken by EPA to control sources of atmospheric emissions of benzene. This report projects atmospheric concentrations of benzene and estimates the number of people exposed to these concentrations. These concentration estimates are developed on an emission source category basis and are then integrated, using population flux estimates, thus deriving total exposure estimates. Activity patterns (community) are taken into account.

U.S. Environmental Protection Agency. The Carcinogenic Assessment Group's Final Risk Assessment on Arsenic. EPA 600/6-81-002, U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards, Washington, D. C., May 1981. 31 pp.

An estimate of the potential cancer hazard to populations exposed to airborne arsenic is given. Data analyzed are from occupational studies, estimates of ambient exposure and the number of cancer cases induced in the general or 'non-industrial' exposed populations. Using a linear model and data from three studies (Pinto *et al*., (1977), Ott *et al*., (1974), and Lee and Fraumeni (1969)), it is estimated that the increase in the lung cancer rate per increase of 1 micrograms/$m^3$ of atmospheric arsenic was 9.4%, 17.0% and 3.3% respectively, with a geometric mean of these values calculated to be 8.1%. Using this value and data from a revised 1979 Stanford Research Institute arsenic exposure assessment, lifetime risk of respiratory cancer to those near cotton gins is calculated to be as high as 0.0134 and higher than 0.00001 for about 2.81 million people due to all sources of air arsenic exposure. Total atmospheric arsenic causing deaths due to respiratory cancer is estimated to be within a 95% confidence interval of 19.4 to 1.2 deaths annually.

U.S. Environmental Protection Agency. The Carcinogenic Assessment Group's Final Report on Population Risk to Ambient Benzene Exposures. EPA-450/5-80-004, U.S. Environmental Protection Agency, Office of Air Quality and Standards, Research Triangle Park, North Carolina, January 10, 1979. 39 pp.

This is one of three reports prepared by EPA to determine what regulatory action should be taken by EPA to control sources of atmospheric emissions of benzene. This report estimates from three studies the leukemia risk associated with current general population exposures to benzene in the United States.

U.S. Environmental Protection Agency. Draft Research Plan for Monitoring Human Exposure to Environmental Pollutants in All Media. Prepared by the Office of Monitoring Systems and Quality Assurance. October 7, 1981.

The goal of this draft Research Plan was to provide a long-range view of the research needs in monitoring human exposure to environmental pollutants. An attempt was made to survey a significant portion of the entire field of monitoring human exposure to all pollutants in all media, with the exception of those pollutants associated with mobile sources; a separate research plan deals with vehicular air pollutants.

U.S. Environmental Protection Agency. Formulation of a Preliminary Assessment of Halogenated Organic Compounds in Man and Environmental Media. EPA-560/13-79-006, U.S. Environmental Protection Agency, Office of Toxic Substances, July 1979. 441 pp.

This comprehensive report covers the information which has been gathered on this program during the past year. A model is developed for making a comparative assessment of halogenated organics in man and environmental media.

U.S. Environmental Protection Agency. Handbook for Performing Exposure Assessments. Compiled by Exposure Assessment Group, Office of Health and Environmental Assessment. September 1981.

This document reviews exposure assessment activities within program offices at EPA and reviews the models and monitoring methods employed in exposure assessment efforts. Methods for dealing with uncertainty in exposure assessment are discussed.

U.S. Environmental Protection Agency and U.S. Department of Energy. Inventory of Current Indoor Air Quality-Related Research. Prepared for the U.S. Environmental Protection Agency Office of Research and Development, Industrial Environmental Research Laboratory, Cincinnati, Ohio, April 1981. 194 pp.

This document summarizes ongoing federally funded research effects aimed at characterizing indoor air quality. Study objectives and funding levels are given.

U.S. Environmental Protection Agency. National Interim Primary Drinking Water Regulations; Control of Trihalomethanes in Drinking Water. 44 Federal Register 68624-68706, November 29, 1979. (40 CFR Part 141)

U.S. Environmental Protection Agency. Potential Worker and Consumer Exposures to Nitrilotriacetic Acid (NTA) in Detergents. EPA-560/11-79-008, Prepared by Midwest Research Institute for the U.S. Environmental Protection Agency, Office of Toxic Substances, June 1979. 40 pp.

U.S. Environmental Protection Agency. A Research Plan for Population Exposure Monitoring Methodology: Vehicular Air Pollutants. Prepared by the Office of Monitoring Systems and Quality Assurance and the Office of Mobile Source Air Pollution Control. November 16, 1981. 89 pp.

U.S. Environmental Protection Agency. Standard Support and Environmental Impact Statement: Emission Standard for Vinyl Chloride. EPA-450/2-75-009, U.S. Environmental Protection Agency, Office of Air and Waste Management, Office of Air Quality Planning and Standards, Research Triangle Park, North Carolina, October 1975, 426 pp.

A national emission standard for vinyl chloride emitted from ethylene dichloride-vinyl chloride and polyvinyl chloride plants is being proposed under the authority of section 112 of the Clean Air Act. Vinyl chloride has been implicated as the causal agent of angiosarcoma and other serious disorders, both carcinogenic and noncarcinogenic, in people with occupational exposure and in animals with experimental exposure to vinyl chloride. Reasonable extrapolations from these findings cause concern that vinyl chloride may cause or contribute to the same or similar disorders at present ambient air levels. The purpose of the proposed standard is to minimize vinyl chloride from all known process and fugitive emission sources in ethylene dichloride-vinyl chloride and polyvinyl chloride plants to the level attainable with best available control technology. This would have the effect of furthering the protection of public health by minimizing the health risks to the people living in the vicinity of these plants and to any additional people who are exposed as a result of new construction. This is estimated to reduce emissions from a typical ethylene dichloride-vinyl chloride plant by approximately 95 percent. Environmental Impact Statements and Inflation Impact Statements quantifying the impacts of the proposed standard and alternative control levels are included in this document.

U.S. Environmental Protection Agency. Study of Children's Blood-Lead Levels Within Families. EPA-600/1-78-067, Health Effects Research Laboratory, Cincinnati, Ohio, November 1978. 135 pp.

Comparative studies of the biological indices of elevated exposure to lead in children and adults were conducted with the intention of reaching a better understanding of lead absorption in children. Three family groups were examined. Group 1 consisted of families who lived in the vicinity of a lead smelter and whose fathers were occupationally highly exposed to lead. Group 2 consisted of families settled in the same area, but whose fathers had no supplemental occupational exposure to lead. The third was the control group consisting of families who lived in an area with very low exposure and whose fathers were not occupationally exposed to lead. Families were selected with one child under 4 years and, if possible, another child of school age. The environmental survey included analyses of lead in air, dust fall, household-dust, and drinking-water. Three biological parameters, erythrocyte-aminolevulinic dehydratase activity, erythrocyte protoporphyrin, and blood lead were determined. On the basis of these parameters the following sequence of decreasing lead absorption was established in family members living in an area with elevated lead exposure: fathers, school-age children, children up to 4 years old, mothers. Children with fathers occupationally exposed to lead had a slight additional lead exposure in comparison with children whose fathers had no supplemental occupational exposure to lead. It was found

that the population leving near a lead smelter, except for fathers occupationally exposed to lead, had biological findings at the level of a "moderately elevated" exposure, while those occupationally exposed had "excessive" exposure.

U.S. Environmental Protection Agency, et al. Summary of the Workshop on Exposure to Environmental Agents, Their Metabolism, and Mechanisms of Toxicity--Research Needs. Prepared by the Project Group on Exposure and Metabolic Mechanisms, Task Force on Environmental Cancer and Heart and Lung Disease. August 1981. 119 pp.

This document summarizes the results of a series of interagency workshops covering a range of topics related to exposure to environmental agents. One of the areas addressed involved methods for monitoring individual human exposures to environmental pollutants. Three recommendations were made in this area. The first was that appropriate government agencies should reexamine their programs in light of the Task Force's belief that monitoring direct human exposure is more valuable than monitoring at fixed outdoor stations and in light of new techniques available. Four types of programs were suggested for consideration -- total exposure studies, combined exposure/dose studies, baseline monitoring, and monitoring in support of epidemiology. The second recommendation was that further advances in sampling and analytical techniques should be pursued in three broad areas: sampling techniques (e.g., development of personal air quality monitors for particulates, polyaromatic hydrocarbons, and fibrous aerosols), analytical techniques (e.g., techniques for analyzing chemicals in food, particularly volatile and non-volatile organics), and microanalytical techniques. The third Task Force recommendation was that a strong quality assurance program be initiated for all government-sponsored collection of environmental data.

U.S. Environmental Protection Agency. VOC Fugitive Emissions in the Synthetic Organic Chemicals Manufacturing Industry--Background Information for Proposed Standards, Draft EIS. EPA-450/3-80-033a, U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards, Research Triangle Park, North Carolina, November 1980.

Standards of performance to control fugitive emissions of VOC from new, modified, and reconstructed Synthetic Organic Chemical Manufacturing Industry (SOCMI) plants are being proposed under section 111 of the Clean Air Act. This document contains information on SOCMI, emission control technology for fugitive emissions of VOC, regulatory alternatives which were considered, analyses of environmental, energy, costs, and other technical data to support the standards of performance.

U.S. General Accounting Office. Indoor Air Pollution: An Emerging Health Problem. Report to the Congress by the Comptroller General. September 24, 1980.

Traditionally it has been presumed that a person was protected from polluted air when indoors. Recent research has shown, however, that this may not always be true. Various harmful pollutants--including radon, formaldehyde, and nitrogen dioxide--have been found in the air in homes, offices, schools, and even in recreational facilities. The problem may even be made worse by Government energy conservation programs which encourage the "buttoning-up" of buildings.

Federal efforts to deal with the problem have been piecemeal, receiving little support primarily because no one Federal agency has responsibility for the problem. Until responsibility is assigned to one agency to oversee Federal efforts, they will continue to be ineffectual.

U.S. Environmental Protection Agency. Benzene Emissions from Benzene Storage Tanks--Background Information for Proposed Standards; Draft EIS.

U.S. Environmental Protection Agency. Benzene Emissions from Ethylbenzene/Styrene Industry--Background Information for Proposed Standards; Draft EIS.

U.S. Environmental Protection Agency. Benzene Emissions from Maleic Anhydride Industry--Background Information for Proposed Standards; Draft EIS.

Varma, M. M. and H. M. Katz. Environmental Impact of Cadmium. Journal of Environmental Health, 40(6):308-314, May-June 1978.

It is important to measure the total burden of the aquatic system in evaluating the biological effect of Cadmium (Cd) in natural water. Solubility of Cd depends on pH and carbonate concentration. Under certain conditions, Cd may be undetectable in the aqueous phase, but may be present in considerably higher concentrations in suspended particles and sediment. In general, U.S. water supplies do not exceed the 10-ppm Cd limit. Though airborne Cd is not an important source of direct human exposure except in certain occupational situations, it can be transported over long distances, and is deposited on soil and water or directly on plants through fallout and rain. Cadmium concentration in uncontaminated soil varies widely depending on the composition of the underlying rock, the amount of organic matter, and the permeability. Addition of Cd to agricultural soil by application of sewage sludge, fertilizer, wastewater effluents, and from airborne fallout is likely to result in increased dietary intake of Cd. Human exposure to Cd is primarily from food and tobacco, with minor contributions from air and drinking water. Cadmium deposits in lungs and is absorbed gastrointestinally. Excretion of absorbed Cd is mainly via urine. In humans without abnormal exposure, the largest concentration of Cd is found in the kidneys (about 1/3 of body burden).

Venkatram, A. On setting Air Quality Criteria. *Water, Air, and Soil Pollution*, 10(4):441-449, November 1978.

Air quality criteria can be related to a simple biological response model based on the pollutant uptake rate and the detoxification rate. The concentration required to produce a specified effect over a given exposure time is represented mathematically and plotted on a log-log graph. The curve produced is termed the biological response. A straight line drawn through exposure times of interest represents the measured maximum concentrations as a function of averaging time. A straight line drawn tangential to the curve satisfies the air quality criteria set by the biological response. By integrating the equations, the cumulative effect can be determined for an air pollution index that is not arbitrary, but based on actual biological response. A proportionality constant and the concentration to which the organism can be exposed to continuously with no adverse effects are necessary for the determination and can be difficult to obtain.

Verberk, M.M. and T.M.L. Scheffers. Tetrachloroethylene in Exhaled Air of Residents Near Dry Cleaning Shops. *Environmental Research*, 21:432-437, 1980.

The use of exhaled tetrachloroethylene as an indicator of exposure in urban residents living near dry cleaning establishments is explored.

Vukovich, F. M., W. D. Bach, Jr. and C. A. Clayton (Research Triangle Institute) Optimum Meteorological and Air Pollution Sampling Network Selection in Cities, Volume I: Theory and Design for St. Louis. EPA-600/4-78-030, U.S. Environmental Protection Agency, Environmental Monitoring and Support Laboratory, Las Vegas, Nevada, June 1978.

Walker, M. V. and C. J. Weschler. Water-Soluble Components of Size-Fractionated Aerosols Collected after Hours in a Modern Office Building. *Environmental Science and Technology*, 14(5):594-597, May 1980.

Size-fractionated aerosol samples were collected after hours in a modern 6-story facility that houses 4,000 employees. The total water-soluble content of the aerosol decreases with increasing particle size, as indicated by conductivity measurements. Sulfate and $NH_4^+$ are chiefly responsible for this trend, while $NO_3^-$, $Cl^-$, and $Na^+$ are fairly evenly distributed throughout the size-fractionated samples. Acid ammonium sulfate accounts for the majority of the water-soluble materials in the submicron aerosol. In this size range, the indoor $SO_4^{-2}$ values are very similar to reported outdoor values. Consistent with the building's air filtration system, in the submicron size range the gross features of the indoor and outdoor aerosols are similar, while differences are more pronounced in the larger size ranges.

Wallace, L. Use of Personal Monitors to Measure Commuter Exposures to Carbon Monoxide in Vehicle Passenger Compartments. paper 79-59.2 presented at 72nd Annual Meeting of APCA, 1979.

Wallace L., and W. Ott. Personal Monitors: A State-of-the-Art Survey. Journal of the Air Pollution Control Association, 32:601-610, 1982.

Recent advances in technology have resulted in the development of a number of small lightweight devices that can be carried by a person throughout the day to record his or her exposure to selected air pollutants. The essential characteristics, advantages, and problems associated with personal monitors are discussed and compared to the characteristics of fixed-station monitoring. Personal monitors generally are superior to fixed monitors for estimating personal exposure, at least for selected air pollutants. They make it possible to collect data on pollutant concentrations that people normally come into contact with throughout their daily activities-indoors, outdoors, and in various modes of transportation. These new instruments ultimately could transform the way in which human exposure to air pollution is estimated.

Wallace, L. and Pellizzari, E.D. Direct Measurements of Volatile Organic Compounds in Breathing Zone Air, Drinking Water, Breath and Blood. Environment International, 8, 1982.

The exposure of 17 college students in Beaumont, TX and Chapel Hill, NC to a number of volatile organic pollutants was assessed by measuring levels of these substances in air, water, blood and expired air. Despite the presence of a highly developed petrochemical industry in Beaumont, no systematic differences in exposure levels were found between the two groups of students.

Wallace, L. Individual Human Exposures to Volatile Organic Compounds Encountered During Normal Daily Activities. Submitted to Environmental Research, 1983.

This study attempted to characterize the exposure of nine New Jersey residents to a number of organic environmental pollutants. Temporal, seasonal, and occupational variations of these chemicals in air, food, drinking water and blood were measured. Important routes of exposure for individual pollutants were identified. Correlations between levels within and between various media were analyzed. A model relating exposure to breath levels was postulated and parameters of the model determined for two chemicals.

Walsh, P. J., E. M. Rupp, D. C. Parzyck, et al. Composite Hazard Index for Assessing Limiting Exposures to Environmental Pollutants: Application Through a Case Study. Environmental Science and Technology, 12(7):802-807, July 1978.

The composite hazard index assessment methodology is applied to Cd releases from a smelter complex in East Helena, Montana. Environmental transport models are used to estimate Cd dispersion through air, concentrations in air, deposition on soil and plant surfaces, uptake by plants, intake by animals, and human intake through inhalation and ingestion. A physiological model is used to estimate Cd uptake and

distribution among human organs. A review of toxicological information on Cd resulted in the selection of 200 µg Cd/g wet wt. kidney cortex as a Cd concentration that should not be exceeded due to health risk. A limiting Cd air concentration is calculated as that air concentration that will limit the accumulation of Cd within the human kidney cortex to < 200 µg/g over a 50-yr. exposure period.

Walsh, P. J., G. G. Killough, and P. S. Rohwer. Composite Hazard Index for Assessing Limiting Exposures to Environmental Pollutants: Formulation and Derivation. Environmental Science and Technology, 12(7):799-802, July 1978.

A hazard index methodology is developed for limiting human exposure to environmental pollutants. Hazard index is defined as Q/QL, where Q is a measure of exposure, such as an organ's burden of a pollutant from all environmental pathways, and QL is a corresponding limit that should not be exceeded because of public health risk. Mathematical formulations relating hazard indices to environmental pollutant concentration are developed for each sampling medium (air, water, food) corresponding to each effluent mode. These formulations are used to derive limiting pollutant concentrations such that the resultant measure of exposure will not exceed the health risk limit. Mathematical formulations for composite hazard indices for multiple pollutants are presented. Composite hazard indices conceptually provide an interface between human exposure assessments and human health risk assessment. Their practical determination requires detailed knowledge of source emission characteristics, environmental transport processes, human metabolism, and biological effects.

Withey, J. R. and B. T. Collins. A Statistical Assessment of the Quantitative Uptake of Vinyl Chloride Monomer from Aqueous Solution. Journal of Toxicology and Environmental Health, 2:311-321, 1976.

The presence of vinyl chloride monomer (VCM) in foodstuffs and its demonstrated carcinogenic potential when administered by the oral route has raised questions concerning the quantitative estimation of the safety of the use of food packaging fabricated from rigid polyvinyl chloride. A statistical model, which was tested by curve-fitting data obtained from an oral uptake study, has been demonstrated to be of predictive value. Ninety-five percent confidence limits were also calculated, and the data from this study were compared with those from a previous gas phase exposure study. It was concluded that if the total daily liquid intake contained 20 ppm of VCM then the area generated under the blood level-time curve, for rats, would be equivalent to an inhalation exposure of about 2 ppm for 24 hours.

Yanagisawa, Y. and H. Nishimura. A Badge-Type Personal Sampler for Measurement of Personal Exposure to $NO_2$ and NO in Ambient Air. Environment International, 8:235-242, 1982.

Yocum, J.E. Indoor-Outdoor Air Quality Relationships. A Critical Review. Journal of the Air Pollution Control Association, 32:500-520, 1982.

A comprehensive review of research in indoor air quality conducted since 1972 is presented. A summary of major research efforts is followed by reviews of research related to individual criteria pollutants ($SO_2$, particulates, CO, NOx, ozone, hydrocarbons and lead) and other pollutants (sulfates and nitrates, carbon dioxide, formaldehyde and radon). The current state of knowledge about indoor-outdoor relationships for these pollutants is summarized.

Ziskind, R., et al. Pilot Field Study: Carbon Monoxide Exposure Monitoring in the General Population. Environment International, 8:283-293, 1982.

A pilot field study was conducted with nine members of the general public to measure carbon monoxide exposure using personal monitors. The principal study objectives were to design and evaluate the research protocol and the instrumentation performance for application to the conduct of a large-scale personal monitoring program. Integrated carbon monoxide exposure was monitored and recorded according to type of activity such as "commuting" or "at work" for approximately 45 days by each subject. All subjects except one were able to handle both the equipment and data recording requirements with no significant problems. The data consisted of 355 person-days each over 6 hours duration, and weekdays only, from which 8 hour average personal exposure levels could be computed. The 9 ppm ambient air quality standard was exceeded on 22 person-days. Elevated carbon monoxide concentrations during the commuting activity were frequently associated with the exceedences.

Zweidinger, R., et al. Monitoring Individual Exposure--Measurements of Volatile Organic Compounds in Breathing-Zone Air, Drinking Water, and Exhaled Breath. U.S. Environmental Protection Agency, Office of Monitoring Systems and Quality Assurance, Office of Research and Development, 1981.